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OTITIC MENINGITIS.*

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It gives me great pleasure to address the Section of Laryngology and Otology of the College of Physicians of Philadelphia on this occasion, and upon a subject which has interested me for many years. It seems rather remarkable that, while intra-cranial complications of middle ear suppuration have been largely amenable to treatment owing to the advances in surgery, otitic meningitis still remains the one complication which to a great degree baffles our skill.

In this connection it might be well to remember the importance of suppurative otitis media in relation to involvement of the intra-cranial structures. A number of years ago (*Medical News*, Oct. 17, 1903) I collated from the reports of the New York Eye and Ear Infirmary 19,000 cases of middle ear suppuration, both acute and chronic. Out of these 19,000 cases of middle ear suppuration one patient in every 88 suffered from some intra-cranial lesion, either an epidural abscess, sinus thrombosis, brain abscess, or meningitis. Fortunately, otitic meningitis is perhaps the rarest intra-cranial complication of middle ear suppuration. Statistics of this kind are rather difficult to collate for the reason that in our hospital statistics, account is only taken of the operative cases, and many cases of otitic meningitis are so hopeless from the moment the diagnosis is made that no operative interference is attempted.

*Read before the Section of Laryngology and Otology of the College of Physicians of Philadelphia, April 17, 1918.

In the first place we have to consider exactly what is meant by the term "Otitic Meningitis." In its broadest sense it covers all inflammations of the coverings of the brain due to a middle ear infection. While one class of meningeal inflammation is a comparatively simple complication and offers practically no menace to life, other classes are always severe and usually terminate fatally. The simplest form of otitic meningitis which presents itself to the otologist is the well known extradural or epidural abscess, consisting of a collection of pus between the dura and the cranial wall. These extradural abscesses occur most frequently in the region of the lateral sinus, usually at the knee, and next most frequently in the middle cranial fossa. They present no symptoms aside from localized headache, local tenderness, sleeplessness, and a slight elevation of temperature. The condition is frequently found only on operation, the symptoms being so slight that the disease escapes recognition until at the time of the mastoid operation pus is found in one of the localities named. By bearing in mind, however, the possibility of such a collection of pus in every case of middle ear suppuration with mastoid involvement, we are frequently able to make a diagnosis. The patient complains ordinarily of sleeplessness, of localized headache over the abscess, and of limited tenderness on percussion. There is also a slight elevation of temperature. Given these symptoms in any case of middle ear suppuration, either acute or chronic, the surgeon should always be on his guard as to the possibility of finding a collection of pus between the dura and the bony wall of the cranial cavity. This represents the simplest form of otitic meningitis.

The examination of the spinal fluid in a case of this kind is interesting, in that ordinarily we have an increase in the globulins, and a moderate increase in the cell count. These changes in the spinal fluid have enabled me in doubtful cases to sometimes make a diagnosis prior to operation.

I am mentioning this comparatively trivial complication because I wish to show that by careful observation we may be able to detect even slight involvement of the meninges. These cases all do well on operation. It is simply necessary to evacuate the abscess thoroughly, expose the entire dural area involved, and pack the abscess cavity with iodoform gauze. As I remember, I have seen but very few cases of this kind result fatally.

In the class of cases already alluded to, the dura is the only covering of the brain involved. The dura, being hard and resistant, offers an almost impenetrable barrier to the entrance of the

pathogenic organisms to the more delicate arachnoid and pia. Those forms of meningitis, which from their gravity chiefly command our attention, are not inflammations affecting the dura, but those which affect the pia and arachnoid space.

The simplest form of meningitis of this character was first described by Quincke under the name of serous meningitis (*Sammlung klin. Vorträge Innere Medicin*, i, 29, p. 653). It must be remembered that this term "Serous Meningitis" was employed before we knew as much about the bacteriological investigation of the spinal fluid as we know at the present time. At present, I am in doubt as to whether the term "Serous Meningitis" should ever be employed. As you will all remember, the disease described by Quincke was characterized by headache, vomiting, muscular rigidity, temperature of 101 to 103, presence of the Kernig sign, occasional choked disc, and by an accumulation of an excessive amount of cerebro-spinal fluid. This fluid was clear on spinal puncture, and was germ free.

Personally, I have always looked upon a serous meningitis either as the first stage of diffuse suppurative meningitis, or as a condition indicative simply of intra-cranial pressure. In support of this latter view we have only to remember those cases where we have all of these symptoms described by Quincke occurring in cases of intra-cranial neoplasms, with the exception that we have no temperature in these latter cases. With these symptoms given in the case of an otitis, I, personally, am rather inclined to look upon them not as cases of meningitis, but as cases in which, due to the inflammatory process in the ear, there has been some blocking of one of the cerebral foramina, thus interfering with the free circulation of the cerebro-spinal fluid. This, I believe, is really the first stage of an otitic meningitis in every instance. I believe also that cases of so-called meningeal irritation and of meningismus described by some writers belong to this category.

Before proceeding to a consideration of the various forms of true otitic lepto-meningitis, I wish to call attention to a series of cases which have undoubtedly caused some confusion, and have been classed as cases of meningitis of otitic origin. I refer to what might be properly called coincident meningitis. In these cases we have the history of an acute otitis media, and laboratory investigation shows the pathogenic organism to be either the streptococcus, or the pneumococcus. In these cases the otitis may for several days run a practically classical course with no symptoms of mastoid involvement other than a profuse discharge. Sud-

denly, meningeal symptoms will appear, and the patient will die as a result of the meningitis. In many of these cases autopsy fails to reveal extension of the disease from the middle ear to the meninges. As we all know that quite a number of cases of pneumococcus meningitis occur with no aural symptoms, I am quite certain that a fairly large proportion of these cases are cases of coincident meningitis; that is, we have a pneumococcus meningitis and, at the same time, a pneumococcus otitis. That the meningitis is the actual result of the otitis in many of these cases is not proven by autopsy.

When we come to a consideration of otitic meningitis, we may divide the disease into three groups; first, the fulminating cases; second, the regular or frank cases; and third, the latent cases. In the fulminating cases there is very little difficulty in making a diagnosis. The onset of the disease is sudden, and is characterized by severe headache, vomiting, and a high temperature ranging between 104 and 105 degrees. Photophobia is usually present, and the pupils are usually contracted. Coincident with these symptoms, we generally have some interference with the motility of the ocular muscles, giving rise to an internal strabismus. This as a rule occurs on the side of the ear involved, but may occur on both sides, or may only occur on the opposite side. Early in the attack muscular rigidity becomes pronounced and, in children, convulsions are exceedingly common. The muscular rigidity usually first involves the muscles of the neck, the head being drawn backward and buried in the pillows. This rigidity gradually extends to all the dorsal muscles, so that even in a few hours after the inception of the attack the patient's entire body may be raised from the bed by putting the hand under the occiput. The muscles of the lower extremities are also involved early, and the Kernig sign is well developed early in the disease. The Babinski sign also makes its appearance early in these fulminating cases. The pulse, in the early stages, is rapid, and the pulse temperature ratio is not disturbed. Delirium comes on early, and is quickly followed by stupor. In severe cases with pronounced delirium, physical restraint of the patient is often necessary. Difficulty in swallowing may come on early, or may be a late symptom. In a typical fulminating case the respiratory symptoms come on early in the disease, and the respiration becomes Cheyne-Stokes. The pulse becomes slow, owing to the increased intra-cranial pressure, and a fatal termination occurs within twelve or forty-eight hours after the inception of the symptoms.

An examination of the ocular fundus will usually reveal the signs of choked disc, either beginning, or fully developed. This sign is of great value as a diagnostic factor, and may be present in all cases of meningitis, no matter how mild the type. An examination of the ocular fundus should always be made in all cases presenting symptoms of intracranial involvement. Unfortunately, the degree of the fundus changes in no way indicate the severity of the meningitis, nor do they determine that this is the intracranial complication. They simply indicate increased intracranial pressure.

The spinal fluid obtained by lumbar puncture in these cases naturally clears up the diagnosis, although this can be made without difficulty, in fulminating cases, upon the symptoms. The spinal fluid is found to be under a great increase in pressure, is turbid, the cell count is greatly increased, globulins are present, and pathogenic organisms are found usually in smears of the fluid, but invariably upon culture. These fulminating cases, while they do occur frequently as the result of an otitis media, either acute or chronic, very frequently are cases of what I have termed coincident meningitis, and they are described in the paragraph immediately preceding. It is in these cases, I think, that an error is apt to be made, not in making the diagnosis of meningitis because the symptoms are so evident that no error can occur here, but in classing all the cases as cases of otitic meningitis.

A case of this character came under my observation not long ago in one of the military hospitals in New York City. The patient entered the hospital with an acute otitis. A free incision of the drum membrane was made, and profuse discharge appeared. The temperature was 104, but gradually fell after incision. No mastoid tenderness was present, no headache, no vomiting, and the X-ray showed the mastoid slightly cloudy. I first saw the patient on the second day of the otitis. The only disturbing feature about the case was the temperature elevation. The temperature gradually fell until it became normal on the fifth day of the otitis. No mastoid tenderness had appeared at any time, and the fundus of the canal, which had been much swollen, began to resume its normal appearance, although at this time there was a great deal of sinking of the upper and posterior wall of the canal. On the evening of the fifth day the man suddenly became delirious, then went into coma. The spinal fluid was under great pressure, the cell count was high, globulins were present, and a subsequent culture showed the pneumococcus. The mastoid was opened. Free pus

was found, but the cell walls were not broken down. No avenue of invasion extending to the meninges was discernable. There had been no nystagmus, and the labyrinth reactions had been normal, showing no evidence of invasion along the internal auditory meatus. A subtemporal decompression was done at the same time with the mastoid operation. The meninges presented the characteristic appearances of acute meningitis. This case was one of coincident meningitis of the fulminating variety, and was not dependent upon the otitis.

Cases of this fulminating type are occasionally seen when the meninges are injured at the time of the mastoid operation. While usually coincident to surgical infection at the time of the operation, I am inclined to class most of these cases as coincident meningitis. I have had only one case in my own practice, and have seen but few in consultation. I know from experience that accidental wounding of the dura is as a rule not followed by symptoms of acute meningitis. In the cases that I have seen, the meningeal symptoms have come on so rapidly after the traumatism that it would be unreasonable to attribute the meningitis to the dural injury.

Another type of meningitis which we have to consider is what may be called cases of frank meningitis. These present absolutely no difficulties in diagnosis. The symptoms are usually headache, vomiting, rapid pulse, a persistent temperature of about 103 or 104, rigidity of the neck, a positive Kernig, and sometimes Babinski. Choked disc may be present. When these cases appear as the result of any invasion through the internal auditory meatus, we ordinarily have symptoms due to the involvement of the internal ear, such as profound or absolute deafness, nystagmus first toward the affected side, but quickly changing to the diseased side as soon as the labyrinth becomes greatly involved. Vertigo is also present in these cases. An early symptom which in my own practice has been of value in the early stages of meningitis where the invasion occurs through the internal auditory meatus is that the direction of the nystagmus can be changed by shaking the head. It has frequently been my experience to have a house surgeon report to me that a patient had a nystagmus in one direction, that is, we will say for example, toward the diseased side. On seeing the patient at the hospital I have quite frequently seen the nystagmus to the opposite side, or possibly none at all. Where there has been no nystagmus, I have resorted to the test of shak-

ing the patient's head somewhat violently. This has resulted in the eliciting of a nystagmus in one or the other direction, frequently in both directions. In cases where nystagmus had been reported to one side, and I have found the nystagmus to be to the opposite side at the time of my visit, this same procedure, that is, shaking the patient's head rather violently, has elicited nystagmus in the other direction. My own idea regarding these cases is that where the invasion of the meninges occurs through the labyrinth, in the first stages of the inflammatory process certain changes occur in the labyrinthine fluid. Owing to these changes the normal mobility of the fluid is altered. In patients with normal labyrinths, the head may be shaken violently, but owing to the free circulation of the labyrinthine fluid, these sudden changes in position of the head do not disturb the labyrinthine equilibrium, and, consequently, do not cause nystagmus. With a change, however, in the character of the labyrinthine fluid, this fluid is not able to move as freely in the semi-circular canals as when it is normal, consequently, sudden changes in the position of the head are followed by nystagmus. These symptoms I have found very valuable in quite a number of cases. It goes without saying, as before stated, that these symptoms will be present only in those cases where the invasion occurs through the labyrinth. Where such invasion occurs either by direct extension into the middle cranial fossa, or in any other region excepting through the labyrinth, this sign will be wanting. It may be argued that the sign just mentioned is really an indication of labyrinthitis rather than of meningitis. I am quite willing to accept this statement, but believe that it is difficult, and perhaps impossible in many cases to draw the line between an invasion of the labyrinth, and an invasion of the meninges. The labyrinthine fluid and the cerebro-spinal fluid are the same, and are in free communication. In other words, a suppurative labyrinthitis frequently represents the first stage of an acute meningitis. The characteristics of the cerebro-spinal fluid in these frank cases of meningitis enable us to easily clinch our diagnosis. The cerebro-spinal fluid is under pressure, is turbid, the cell count is high, the globulins are increased, and the fluid may show the presence of pathogenic organisms either upon smear or cultivation. In this class of cases also, there is no difficulty in making a diagnosis. Where the invasion occurs through the labyrinth, the caloric test will show in the early stages a labyrinth which responds slowly to the caloric test, and very soon will show the labyrinth to be absolutely dead

This test is of value, naturally, only when the invasion occurs through the labyrinth.

Another series of cases which present themselves may be termed cases of latent meningitis. These cases I have seen chiefly following operation upon the middle ear and mastoid. The first symptoms which present themselves are those of general malaise, moderate temperature ranging from 99 to 101, some headache, although this is by no means constant and is seldom severe, and vomiting. The vomiting is perhaps the most constant symptom, and is frequently considered to be the vomiting incident upon the administration of the anesthetic. The mere fact that the patient has been subjected to a general anesthetic makes one attach less importance to the vomiting than would otherwise be done. I always regard with suspicion any case of middle ear suppuration operated upon where the vomiting persists longer than one would ordinarily expect after the administration of a general anesthetic, or where this vomiting is more severe than usual. With this vomiting there may be some rigidity of the neck muscles, although this may be very slight, so slight as to escape observation. The Kernig sign in these cases is doubtful, there is no delirium, the mentality is clear, and the patients do not seem to be very ill. If invasion has occurred through the labyrinth, the signs already mentioned in the frank cases may be present, that is, nystagmus and a dead labyrinth. Frequently, however, these signs are wanting. This latent condition may persist for four, five or six days, the temperature fluctuating between 99 and 101½ and 102, the general condition of the patient not changing much, and the patient not seeming to be very ill, yet not reacting from their operative procedure in the characteristic normal manner. The cerebrospinal fluid may be under pressure. The globulins are apt to be increased, but as the globulin test is apt to be positive in all cases where the dura is exposed either by disease or by operation, this fact is not of great value in diagnosis. The cell count is ordinarily increased in these latent cases but may not be very high, frequently being less than 100. It is this latent stage which perhaps offers the best period for operative intervention and, consequently, its recognition is of extreme importance. I have not mentioned here the ocular paralysis because this may or may not occur. If this does occur, it naturally aids in the diagnosis. The differential blood count is of great value, as we often have a high polymorphonuclear count with a high white cell count. The white cell count may be 20,000 or 25,000, while the polymorphonuclear

percentage may be 90. The differential blood count here is of material aid in the diagnosis.

In speaking of the latent cases, one case which came under my observation is particularly interesting. This patient entered the hospital with an acute mastoiditis. At the time of operation a small brain abscess was found, and was drained. The patient had all the symptoms of meningitis, but these subsequently subsided; the temperature became normal, and the patient was up and about the hospital ward. He remained in the hospital for three months. During this time, after the acute symptoms had passed away, the temperature was never above 100, and usually about 99. He presented absolutely no symptoms excepting that he was weak, and inclined to be lachrymose, and also apprehensive as to his condition. The patient, while about the ward, was able to perform light duties, and was only kept in the hospital because each examination of the spinal fluid showed the presence of streptococci. In the absence of any other untoward symptoms, the man was simply kept under observation. He remained in the hospital, as I say, for about three months, when suddenly the temperature rose to 104 or 105, he became comatose and died. At the autopsy the sphenoidal sinus was found to be filled with pus, and it seemed quite possible that the meningitis which caused his death had come through the sphenodial suppuration which had given rise to no symptoms. I mention this case simply to show that a patient may suffer from a suppurative meningitis for months, and that the only evidence that we have of this may be the presence of pathogenic organisms in the spinal fluid. Had it not been for the presence of these organisms this patient would have been discharged from the hospital. This case perhaps demonstrates as clearly as possible that a meningitis may be latent, and may give rise to no symptoms for a considerable period of time.

When we come to the treatment of the condition, we are confronted by the lamentable fact that in a certain number of cases treatment is without avail. I know that this statement will be criticized by many of my hearers who will think that I should say that in all cases of meningitis treatment is without avail. This I am certain is not the case. In an article read before the International Otological Congress in Budapest in 1909, I was asked to consider the treatment of otitis meningitis. One of my assistants, on my request, collated from literature 101 cases of otitic meningitis, and these together with my own formed the basis of this study. I was surprised to find out how many apparently authentic

recoveries from otitic meningitis were recorded in literature. Of the 101 cases collated at that time, 45 were cured, and 56 died. Of the cured cases 34 were cases of so-called serous meningitis, 4 were cases of circumscribed purulent meningitis with serious meningitis, 4 were cases of circumscribed purulent meningitis, and 3 were cases of diffuse purulent meningitis.

The first and foremost indication in the treatment of the condition is to clear up the primary focus of infection; that is, either to do a complete mastoid operation in cases of acute mastoiditis, or a complete radical operation in cases of chronic middle ear suppuration. At the time of such an operation a large area of dura should always be exposed. I am well aware of the fact that experiments seem to show that no decompression effect is secured by simple exposure of the dura, that is the dura seems to be experimentally a perfectly inelastic membrane.

In one case coming under my observation at St. Luke's Hospital a number of years ago, however, a child suffering from double acute middle ear suppuration presented all the symptoms of meningitis. The temperature was high, the neck was stiff, the Kernig sign was present, and the child was absolutely blind. Both optic discs were choked. In this case I did the ordinary mastoid operation, and exposed a large area of dura in the middle cranial fossa and also in the cerebellar fossa on each side. I had intended opening the dura, but decided not to do so at that time, but fully intended to do so on the following day or two days after if the symptoms did not improve. This child made an absolutely perfect recovery with recovery of eye sight. The only other treatment was one or possibly two lumbar punctures. In this case no organism was found in the spinal fluid.

In a second case coming under my observation about two years ago on my Service at St. Luke's, a boy was admitted to the ward with a temperature of 105, stiff neck, Kernig present, photophobia, vomiting, and intense headache. The cell count of the spinal fluid was over 1,000. In this case I directed one of my assistants to perform the mastoid operation and to expose a large area of dura in the middle fossa; that is, to extend the mastoid wound so as to do a subtemporal decompression, but without opening the dura. In this case repeated lumbar punctures were made, and this patient made a complete recovery. Pneumococci were found in the spinal fluid.

I believe, therefore, that two of the simplest measures in treating these cases are first, a complete operation to eliminate the

primary focus with exposure of a large area of dura, and second, repeated lumbar punctures. There is no question in my mind that the relief of the intracranial tension by lumbar puncture acts as a curative measure in a certain proportion of these cases, and that it should be looked upon as a therapeutic measure as well as a very important diagnostic measure.

One of the earliest cases of meningitis that I ever operated upon was treated by division of the dura, and drainage of the subdural space. This patient made a complete recovery, and was well many years after the operation. Crockett, of Boston, has reported a number of cases in which division of the dura and drainage of the subdural space has been followed by cure, so that I believe that a subtemporal decompression may be looked upon as a valuable therapeutic measure in these cases.

My own plan of procedure is not to do a true decompression operation unless the symptoms are extremely severe; that is, unless we are dealing with a fulminating or a frank case. I believe better results would be obtained by clearing up the original focus of suppuration, and exposing a large area of dura at the first operation, and a subsequent division of the dura if the symptoms persist. The slight amount of relief of tension which we obtain simply by exposing the dura may, in addition with lumbar puncture, be sufficient to relieve the symptoms.

In cases where the invasion of the meninges occurs through the labyrinth, as evidenced by the symptoms detailed in the earlier part of this paper, the extirpation of the labyrinth and the drainage of the subdural space in the immediate vicinity of the internal auditory meatus is probably the ideal procedure. This was first advocated by Neumann of Vienna, and, while the number of cures is not large, a certain number of cases have been cured by this measure. I, myself, have had successful cases of this kind, although I am sorry to say they have been few in number.

An interesting modification of this procedure was employed in a case last summer. This patient was suffering from a typical otitic meningitis of the frank variety. Owing to my absence from town, I asked one of my assistants to operate upon the case. The typical Neumann operation was done, and the dura incised at the internal auditory meatus. The dura in the middle cranial fossa was also divided so that a large area of the brain was exposed in the middle fossa. I saw the patient forty-eight hours after the operation at the first dressing. The temperature was about 104, the neck was rigid, Kernig sign was present, the patient was

mildly delirious, and presented all the symptoms of frank meningitis. The spinal fluid contained streptococci. Considerable prolapse of the brain substance was present. I suggested to Dr. Perkins, who had been kind enough to operate on the case for me, the advisability of introducing Dakin's solution into the wound, and into the subdural space. This was done by means of tubes, and this patient went on to complete recovery as we thought. About six months later he re-entered the hospital with some discharge from the ear. He had headache, some rigidity of the neck, and a high temperature. Lumbar puncture was performed, the fluid being under pressure. The temperature fell in a few days, and the patient seemed better. His acute symptoms, however, reappeared later, and he finally died of acute meningitis. The family refused any further operative interferance. Here we have a case remaining well for six months after the operation. I think there can be no doubt that the fatal attack which caused his death was due to a reinfection of the meninges, as the discharge from the ear had persisted since the first operation.

Considerable interest was excited several years ago at the proposal to drain the cisterna magna in cases of meningitis. This procedure was first advocated by Haynes of New York. It seemed logical at first that the best way of attacking this disease would be to drain this great lymphatic space at the base of the brain. Unfortunately, however, this operation has met with very little success. I, myself, have employed it several times, and, while in several cases it has prolonged the life of the patient, I do not know of a single instance in which it has been followed by a permanent cure.

So far, then, as operative interference is concerned, I think we may sum up the facts as follows:

(1) Removal of the primary focus of infection,

(2) Exposure of large area of dura with subdural drainage in cases of the fulminating type,

(3 Repeated lumbar punctures in all cases.

The Neumann operation with incision of the dura at the internal auditory meatus should be employed in those cases where invasion occurs through the labyrinth, but only in those cases. Combined with the Neumann operation more extensive decompression may be necessary, and the use of Dakin's solution in the wound may, I think, be of benefit in a certain proportion of cases.

I have spoken so far simply of operative measures. All efforts to treat cases of this character either by the intravenous injection

of serum or the intraspinal injection of serum have been valueless. I have given this plan of treatment a thorough trial, but every case has proven fatal. The same may be said of the use of urotropin in the spinal canal. Successful results have been reported, but in my own hands this treatment has also been absolutely valueless. Whether or not urotropin administered internally is of the slightest value I am unable to say. I have used it in some of my cases, and some of these cases have done well. As they have all been cases operated upon, however, I do not know how much of the benefit can be attributed to the drug.

I have tried to put before you in these remarks my own position in regard to otitic meningitis. What I would like to emphasize particularly is that while the disease is always grave and frequently fatal, it is not invariably fatal. I believe that we shall save a certain proportion of cases if we insist upon operative interference in every case of otitic meningitis. It is, therefore, necessary to differentiate between true otitic meningitis and meningitis coincident with an otitis. If we do not operate in cases of otitic meningitis, we know that the patient will almost certainly die. As there is some chance of saving life by operation, I believe that operative measures should be employed in every instance, except perhaps in fulminating cases seen very late. Even here we may do something possibly. What I wish to impress upon you is that none of these cases are hopeless unless they are actually *in extremis* when seen by the surgeon.

15 East Fifty-third St.

Carcinoma of the Larynx. R. H. MEADE, *Jour. Missouri State Med. Assn.*, Jan., 1918.

Intrinsic cancer is slow to invade hyaline cartilage and therefore should be operated by some of the more conservative methods which give as good results as laryngectomy and do not leave much deformity. Any case requiring complete laryngectomy is not properly operated if the glands of the neck are left. Extrinsic cancer of the larynx of an advanced stage are not hopeless, as proven by the case described by the author, and last and most important ,early diagnosis is the key-note to successful results. ED.

EAR PROTECTORS.*

DR. CHARLES W. RICHARDSON, Washington, D. C.

The subject of a proper ear protector, to prevent injury to the conductive and receptive apparatus of the auditory mechanism, that will be serviceable under all forms of gun-fire has been the quest of military surgeons for several years. At the inception of the present war, July 30, 1914, experimentors in none of the belligerent countries had perfected an efficient ear protector.

Several devices had been placed upon the market, and had the approval to a greater or less extent among the lay members of the military departments of the various nations, but none of these had received the indorsement of the Medical Military Department.

In the Navy, where the conditions were more acute, on account of turret firing of great guns, the medical officers and the gun crew had practically given up the use of patented devices and depended almost alone on the efficacy of dry or moist absorbent cotton.

Since the great increase in number of those permanently disabled as result of the handicap of loss of hearing, otologists in the service in all countries have sought more diligently, have experimented more actively, to find a device that will be safe, efficient and not impair the hearing too greatly, in preventing injury to the conductive and receptive apparatus, and that may be worn with comfort.

Soon after its formation the Otolaryngological Section of the Division of Surgery of the Head took up the consideration of this subject. The Research Committee of the National Council of Defense has also been doing some experimental work along the same line.

When I came into the Surgeon General's Office, October 27, 1918, this subject was brought immediately to my attention by the expression of a wish that some expedition be made in accomplishing results.

Obtaining from the Research Committee the material which they had in hand, as well as the co-operation of Surgeon G. E. Tribble of the Navy, and Dr. Stacey Guild of the University of Michigan, we proceeded with a series of experiments the results of which seem to give us fair ground to believe we had found at last a fairly

*Read before a joint session of the American Otological Society and the American L. R. & O. Society.

serviceable protector that filled as nearly as possible, all the requirements.

It is interesting to note that all the devices experimented with gave the same actual results as to gradation in the test made physical upon animals and in field work with soldiers.

The results of the test which were first received were those made upon the conducting apparatus. As is well known, these conditions are visible to ordinary vision and the deviation from the normal may be easily noted.

The results of physical experimentation were also easy to be obtained and quickly noted.

The results of the effect on the labyrinth in connection with these experiments, requires quite an elaborate technique in preparing, and considerable time is required in passing the specimens through the successive stages before they can be mounted and examined.

It would hardly benefit us or elucidate the consideration of the subject by entering into the discussion of the question whether the casualties which result in marked impairment to complete loss of hearing is the result of the transmission of the concussion wave through the auditory conductive apparatus, or in part or entirely due to what we in otology denote as bone conduction. We are all aware of the fact that from our earliest days in the study of otology, we have absorbed and taken unto ourselves as an auditory truism that in all forms of violence to the auditory apparatus, excepting fracture of the skull through the temporal bones, that the expenditure of the violence on the conducting apparatus spared the labyrinth.

Nevertheless, after reading the reports of the various experiments and findings of Witt Maack, Von Eiken, Hoessli and others, we are fairly convinced that laboratory findings cannot completely fit in with certain clinical evidences as found in connection with cases subjected to sudden and intense changes in atmospheric pressure. Indeed, it is practically impossible to reproduce the physical atmospheric concussion typified by the bursting of a high explosive shell, in experimental work.

We may so nearly approach the actual atmospheric changes produced by the bursting of a high explosive shell in our experimentation as to be able to draw deductions therefrom as to what in all probability must take place in the actual, but I am very much disinclined to accept them as an accepted result, which seems to be the case with Seibenmann and others.

It seems to me that we will be able to reach results earlier and with more exactitude if we are willing to adopt the belief that air and bone conduction are both active in bringing about the changes in the labyrinth in these disturbances.

Those cases characterized as "shell shock" are the ones in which the element of bone conduction seems to play the most important role. Here we have a most intense, sudden and violent vibration of the atmosphere produced by the detonation of a single highly explosive shell. We have not only the concussion impression made upon the auditory apparatus, but also upon other organs within the body and not in relation with the external atmosphere.

There is a large group of cases, properly designated as concussion deafness, in which the element of aerial conduction plays th all important part. Bone conduction enters only to a minor degree in the development of these cases.

These cases are those which are attended with marked impairment to complete loss of hearing. Very few of these cases show any tendency to improvement or to the restoration of their hearing.

They are mostly due to the prolonged exposure to highly explosive shells, shrapnel, grenades and the constant rattle of machine guns.

Then we have a fairly large group of cases with marked impairment of hearing, quite often complete deafness, in which aerial conduction is the predominating, if not, the only factor.

These types are exemplified by the deafness which is common among gunners in the Navy, artillerymen in the Army, and in other arms of the service where the men have been under small arm fire from the enemy and close to their own artillery fires.

The pathology of these cases is one of uncertainty as present in the human beings, as there has not up to recent publication, been sufficient reports of autopsy findings to warrant us coming to any exact conclusion.

We have a fair amount of material from experimental work, and if we may draw conclusions through analogous condition, nearly approached through physical experimentation, we should have some valuable deductions.

Yohsis's reports of labyrinth sections made after single detonations, in which animals were killed on successive days up to the thirtieth day, show marked changes in the labyrinthian canal. These changes were most markedly manifested in animals killed on the third day after exposure.

In these cases there were marked changes in the cochlea contents. The inner and outer hair cells became greatly swollen. They had

lost their characteristic form and structure, and in part were loosened from their support. The nuclei were displaced upward and in many instances broken into small fragments. Deiters' cells lose their structure and become changed into a homogenous mass. The pillars remain, but are bent and are stated to be indistinct. The narrow tunnel space is filled with a homogeneous material. The outer tunnel and Nuel's space exist no more. The cells of Hensen are deformed, flattened out. These changes are most manifest in the first whorl and in the space characterized by the transition from the first to the second whorl of the cochlea. In successive periods of lengthened intervals in the killing of the animals, we find regeneration gradually taking place, until after a month's time, the regeneration is nearly complete.

The report of the result of autopsy findings in the labyrinth examination in soldiers dead of shell shock, although few yet in number, in a degree confirm the findings in animal experimentation.

J. S. Fraser, Hysterical Deafness in Soldiers, *Lancet*, 1917. II, 872, and in *British Journal of Laryngology, Rhinology and Otology* (November and December, 1917) shows that out of four cases of explosion injury of the ear microscopically examined, (1) rupture of the delicate neuro-epithelial sacs and tubes of the membranous labyrinth was not found in any case. (2) Hemorrhage into the cochlear nerve at the fundus of the internal meatus was present in three out of four cases; such change is quite capable of producing deafness. (3) An early stage of degenerative neuritis was probably present in one case.

In a careful survey of the literature upon shell-shock deafness, I have been impressed with what seems to me a too great leaning to the acceptation that these cases were hysterical in character, without organic changes in the labyrinth. I am willing to accept that some cases of shell-shock are hysterical, without organic change, a synapse, between the cerebral center and the peripheral receptive auditory apparatus; but nevertheless there must be a large percentage which must show more or less organic change within the organ of Corti, temporary to be sure, which recover their function possibly quite suddenly, with the complete regeneration of the temporary labyrinthian changes.

As we have stated before, as these cases seem to be more in the nature of direct concussion rather than transmission through the auditory conducting apparatus, it is very doubtful whether any type of protector will be other than contributory in affording relief.

In the other types of injury to the ear, in which the auditory conducting apparatus plays such an important role, it is essential that we have some device that will arrest the force of concussion, diminish or arrest the injury and will still enable the wearer to hear the voice without any marked impairment of its intensity. Dr. Stacey Guild, who had already started with a series of well calculated experiments for the Committee on Research, National Council of Defense, kindly devoted his work to the services of the Section of Otolaryngology, Division of Surgery of the Head, Surgeon's General's Office. These experiments were made upon guinea pigs to determine—

1. The amount of injury to the conductive apparatus of the unprotected ear.

2. The amount of injury to the conductive apparatus of the protected ear by the several types of antiphons employed.

3. The amount of change produced in the labyrinth of those ears protected by the various types of defenders.

A second series of experiments were made for the purpose of testing the amount of concussion the various types of protectors allowed to pass to the membrana tympani. All these experiments were most ingeniously made by Dr. Stacey Guild, and I do not propose to give more than his result herewith, as he should have all the honor and applause which comes with original work well done.

It is sufficient to state that the various types of protectors, under the various experiments and physical tests, demonstrated the same gradation of protection or efficiency.

The results of the labyrinth examinations have not as yet been received from Dr. Stacey Guild, but I have from him a letter in which he states that the results of his examination show the same relative protection to the labyrinth as the same protectors showed in the protection of the conductive apparatus.

The experiments resorted to by Dr. Stacey Guild of Ann Arbor, Michigan, and the results obtained are displayed in the table which I have placed upon the wall and shall now proceed to explain.

"The fifth table" contains the experimental work with the physical tests of the various antiphons in indicating how much sound concussion they permit to pass. This was indicated by markings on a tambour. The passage of sound waves was indicated in the exact ratio as the experiments to the defenders, in chart No. 4. The position is not altered in any particular.

The report, which was furnished by the Otolaryngological Section of the Division of Surgery of the Head to the Surgeon General, we

are able to present to you. This will furnish as the conclusions made through our various forms of investigations.

MEMORANDUM FOR THE SURGEON GENERAL:

Subject: Ear protectors for the benefit of the soldiers in actual conflict.

1. The subject of protection to the soldier at the front, from the various forms of injury to the auditory apparatus, or to its receptive center, is so important that it is imperative that this department should suggest to the non-medical divisions of the Army what forms of device will lessen the number of those who suffer the ill consequences of injury to the auditory apparatus.

There are various points of view from which to consider this subject.

First. There is the immediate incapacity of the affected man for service, for a shorter or longer period, some of whom must be discharged.

Second. As all men who are subject to injury of the auditory apparatus require medical attention, the time of medical officers, nurses and equipment should be available for other purposes.

Third. The more severe cases result in permanent deafness, which, besides being a great handicap, impairs the soldier's later social and industrial life.

Fourth. The subject of pension and compensation becomes an immediate financial consideration.

2. The *causes* of injuries may be divided into two different groups:

First. Those which are due to a single detonation, or continued single detonations, such as artillery fire; and,

Second. Those which are due to continuous sounds such as continued concussion of the air as produced by massed artillery and trench mortars and machine guns.

3. The *character* of the injury may be divided into three different groups:

First. Rupture of the membrana tympani and other injuries of the conductive apparatus.

Second. Those caused by organic injury from slight to complete destruction of the labyrinth.

Third. A large number of cases which do not belong to either of these groups, but in which both may be factors; functional disorders most frequently of the central nervous system.

4. It has been the object of the Section of Otolaryngology to make a thorough and careful investigation of the various forms of devices that have been invented for the purpose of lessening these various disturbances. All of these have one single object: that is, the lessening of the severity of the concussion impact, either of the single or continuous type at its receptive point, the conducting apparatus of the ear. These devices have as their primary conception that the injury produced by air concussion from detonation is transmitted through the conducting apparatus. All of them have the mechanical idea of lessening this condition by shutting off in various ways, the force of the air concussion, and yet permitting sound waves to reach the membrana tympani so that the soldier may be protected, yet hear. Necessarily, most of these appliances diminish to a certain extent the hearing power. These mechanical devices have been tested on the living animal; have been tested physically to show how much each one permits the passage of the force of air concussion to the membrana tympani, and also rather imperfectly on the human subject at such stations as Indian Head and the Navy Yard at Washington.

5. It is rather interesting and singular to note that all three types of experiments actually coincide as to the gradation of each instrument. Before going further, I want to state that a great deal of honor is due to Dr. Stacey Guild, of Ann Arbor, Michigan, for his experimentation, physical and upon animals. He not only studied out the types of experimentation to be made, but he also conducted them so thoroughly and satisfactorily that there can be no question as to their character. It has been through his experimentation that results have been reached. Dr. Guild has other experiments, partially completed,

to bring before us, which will show much as to the types of injury produced by animal experimentation upon the labyrinth. But we cannot wait longer for these experiments, as the time is now coming closely to hand when the soldier will need the assistance of these various mechanical devices. We have sufficient data, we feel, to decide the question.

It will hardly be necessary to go into the various types of experimentation, and the lines along which they are carried out, as it will take a good deal of your time, and while interesting, would not enable you to come to a quicker conclusion. I simply present to you the results:

6. These are three important features in the device:
   First. Applicability.
   Second. Safety.
   Third. Cheapness.

All types of hard and metallic forms of protectors are dangerous, because in cases of gunshot wounds, shrapnel wounds about the auricle or canal, they are likely to become secondary foriegn bodies. Therefore, we feel very much inclined to eliminate mechanical devices, such as the Wilson-Michaelson and the Mallock-Armstrong. Some of the cheapest, while fairly good protectors, should be ruled out, because they cut off the conduction of air sounds too greatly.

There is no question, through all of our experimentations, but that we have found one actual protector in what is known as the British Tommy, manufactured by George F. Berry, 4 Cullum street, Fenchurch St., London, E.C. This device is simple, easy to introduce, and causes no undue pressure, and is easy to remove. While it cuts down the hearing, it does not cut it down sufficiently to impair the voice beyond military needs. It prevents impact of concussion upon the membrana tympani, the conducting apparatus; it is safe; there is no possibility of forcing it in against the membrana tympani; it is not likely to be any more conducive to secondary foreign bodies than anything that could be worn in the war; and it is comparatively cheap. Attached find copy of contract which Mr. Berry was willing to make for this government. In all of our tests, as you will observe by the charts and exhibits which accompany this paper, it has proved itself the best protector. Actual experimentation upon the living has been impossible with the Tommy at our station here, because we have not been able to procure these protectors. The one set which I transmit with this communication is the only pair which we have. The cost is about a shilling a pair; they can be procured in London, and furnished to our troops on the western front.

The next most satisfactory is the Mallock-Armstrong, made by the Mallock-Armstrong Defender Company, Palmer St., Westminister, London, S. W.

The only objection we have to the Mallock-Armstrong is the fact that it is made out of hard rubber, and is apt to become secondary point of foreign body injuries. In other respects it is nearly as good as the Tommy. It is not as easy to introduce as the former device.

The next device which I would like to call your attention to is the Baum. It is very simple; very easily introduced into the ear; not so easily removed. It can be worn for longer or shorter period without causing any inconvenience to the patient. It is light in weight, and there are practically no dangers attending upon its wearing as regards secondary foreign bodies. It is not nearly as good as the other two mentioned. It does not present the fine degree of prevention as do the other to concussion impacts; but it is an American invention, and can be bought at a very reasonable price.

I cannot close without referring to the Wilson Michelson device, which is planned somewhat on the type of the Mallock-Armstrong. It has a movable valve, which has been demonstrated under our experimentation not to move as freely as it should with the detonations with which we have experimented. Under more forceful concussion, such as takes place in actual warfare, it might respond more favorable. It has the advantage of being a perfect conductor for the voice. It has

the disadvantage of being made of hard rubber, and therefore possesses the danger of secondary foreign body injury. It is from experimentation, a little difficult to wear, and for longer wear it would be inconvenient.

Cotton, saturated with glycerine or vaseline is the cheapest of all; most available, easy to obtain, constantly at hand. It is practically within the reach of every soldier. Soldiers are very much inclined to use cotton in the dry state. It is only in the wet state that it is of any value to prevent shock concussion. This wetting should be done preferably with glycerine or with vaseline. Either one impairs the conduction of sonorous sound waves. Therefore, while it is the cheapest, the most easily available, one that is most likely to be used, it has the disadvantage of deafening the wearer more than any other.

7. In closing, I would suggest that some arrangements be made by which several thousand of the Tommy could be purchased, and be placed in the hands of the troops in the field.

I would also suggest that rules be made by which the soldiers to whom these apparatus are given, be commanded to wear them, the same as they are with their gas masks.

I will also suggest that if any other forms of mechanical apparatus or of cotton saturated, is thought of being used, that it be bought in the same quantity, and on the same conditions as the Tommy, so that we can have definite, actual warfare experiments as to which mechanical device, or cotton is of the greatest value.

CHARLES W. RICHARDSON,
Major, Medical Reserve Corps.
In charge of Section of Otolaryngology.

ADDENDUM TO MEMORANDUM FOR THE SURGEON GENERAL:

(Re: Ear Protectors for the benefit of soldiers in actual conflict.)

Since the completion of the preceding report some new examinations of the labyrinthine injuries in experimental cases have been obtained, and they might slightly modify our report with regard to the other experimental findings.

As is well known, injuries to the membrana tympani often give relief to the labyrinthine contents. In other words where the force of the concussion is expended on the conducting apparatus, it relieves the impact on the internal ear or labyrinthine structures, and vise versa.

While it is possible that there may be an alteration in our report, when the full result of labyrinthine tests come in, we are not inclined to believe that they will affect the result of our original experiments.

It is well to call attention to the fact that there may be, as there has been, some objection to the wearing of rubber in the auditory canal. As this wearing is not continuous we do not think it will militate against the value of the rubber instrument that we have suggested. Of course, the simpler type of instrument which produces no pressure would be of the utmost value; but on the other hand, they do not protect.

MEMORANDUM FOR THE SURGEON GENERAL:

1. The summary of Major C. W. Richardson's report on Ear Protectors is as follows:

1. Of the four protectors tested, the British Tommy is the best.

2. Soldiers are in the habit of using dry cotton as a protector. Cotton is efficient only when moistened with glycerine or vaseline. It deafens the wearer more than the Tommy.

3. It is recommended that several thousand of the Tommy protectors be purchased and issued to the troops with orders to wear them the same as their gas masks.

4. It is recommended also that cotton saturated with glycerine and vaseline be issued to a certain number of men, so that the relative merits of the Tommy and vaseline cotton can be determined, and the question as to which is the more practical under the conditions of actual warfare.

HARRIS P. MOSHER,
Major, Medial Reserve Corps.

TABLE I.

Control Emperiments.

Animals with both ears open; no apparatus in either ear.

Guinea pig No.	Distance of nearest ear from muzzle.	No. of shots fired.	Side.	Condition of tympanic membrane at autopsy.	Amount of coagulate in and on mucous membrane of the middle ear.	Evidence indicating edema of the mucous membrane.	Remarks.
45	60 cm.	1	Rt.	Intact—radial streak of coagulate	Small	Slightly loosened	Shooting from above and in front of animal downward at 20° angle with area
			Left	Intact	One large the horizontal	Slightly loosened	
44	30 cm.	1	Rt.	About ⅓ of area broken	Medium	Loosened	Shooting from above and in front downward about 20° slightly along left side
			Left	Whole center gone	Small	Loosened	
16	15 cm.	1	Rt.	Off along most of circumference	Medium	.....	From above and behind directly over animal
			Left	Not quite as bad as rt. side	Medium	Loosened	
17	15 cm.	5	Rt.	Intact	None	None	(See text matter below) From above and in front along rt. side of animal
			Left	Completely gone	Large	.....	
18	5 cm.	3	Rt.			None	(See text matter below) From above and in front along left side.
			Left			.....	
39	10 cm.	2	Rt.	Off of ¾ of circumference	Medium	Loosened	Shooting from directly abve
			Left	Fringes only left	Large	Loosened	

The finding of normal middle ear parts in one side of No. 17 and No. 18, after shots fired at so short a distance is to be interpreted only on the basis of the external canal having been pinched shut by the jaw pieces of the holding apparatus. These cases were among the first animals used and thereafter special precaution was taken to prevent repetition of this condition, as has been mentioned in the description of the routine procedure. For a short time after the shooting the animals appeared to be somewhat dazed, but none were unconscious when released. The last one listed above was not placed in the holding apparatus and it was able to run away after each shot; it did not stagger, but seemed somewhat dazed.

TABLE II.
Control Experiments.
Animals with glass tube and rubber "ear" (without any preventive device inserted) in one ear and the other ear open.

Guinea pig No.	Side	Smallest diameter of the glass tube in mm.	Condition of wax packing after shooting.	Condition of tympanic membrane at autopsy.	Amount of coagulate in and on mucous membrane of the middle ear.	Evidence indicating edema of the mucous membrane.	Remarks.
19	Rt.	3.5	OK	Ruptured badly	Medium	.....	Two shots—one at 10 cm. from each ear backward along sides.
	Left	....	.....	Ruptured badly	Medium	.....	
52	Rt.	....	.....	Off on ¾ of circumf.	Large	Slightly loosened	Usual arrangement*
	Left	3.5	OK	Fringes	Large	Badly loosened	
51	Rt.	....	.....	Fringe	Large	Slightly loosened	Usual arrangement*
	Left	3.1	OK	Fringe	Large	Slightly loosened	
50	Rt.	....	.....	Fringe	Large	Loosened	Usual arrangement*
	Left	2.5	OK	Fringe	Large	Loosened	
49	Rt.	....	.....	Off on ⅛ of circumf.	Medium	Loosened	Usual arrangement*
	Left	2.0	OK	Off on ½ of circumf.	Medium	Badly loosened	
47	Rt.	....	.....	Off on ⅛ of circumf.	Medium	Loosened	Usual arrangement*
	Left	2.0	End of glass partly stopped	Intact	Streak about tympanic ring	None	
42	Rt.	....	.....	Fringe	Medium	None	Usual arrangement*
	Left	2.0	End of glass partly stopped	Intact	Small	None	
43	Rt.	....	.....	Fringes	Medium	Slightly loosened	Usual arrangement*
	Left	1.5	OK	Intact	None	None	

*One shot at 15 cm. fired with the muzzle of the pistol in front of and slightly above the animal and pointing downward along the left side at an angle of about 20° with the horizontal, just avoiding the holding apparatus with the bullet.

TABLE III.

Record of the Unprotected Ear (Control Ear) of the Animals with which the other ear was "protected."

(The right ear was open in all cases.)

(For the shooting arrangement, see footnote to Table II.)

Guinea pig No.	Condition of Tympanic membrane at autopsy.	Amount of Coagulate in and on mucous membrane of the middle ear.	Evidence indicating edema of the mucous membrane.
21	Off on ½ circumf.	Medium	.....
22	Off on ¾ circumf.	Small	.....
23	Off on ⅘ circumf.	Small	Loosened
24	Off on ⅓ circumf.	Large	.....
25	Off on ½ circumf.	Large	Loosened
26	Off on ½ circumf.	Medium	.....
27	Off on ½ circumf.	Medium	.....
28.	Only fringes left	Medium	.....
29	¼ area gone	Very small	.....
30	½ area gone	Small	.....
31	Only fringes left	Large	.....
32	Only fringes left	Large	.....
33	Only fringes left	Small	.....
34	Only fringes left	Large	Loosened
35	Only fringes left	Small	None
36	Off on ½ circumf.	Large	Loosened
37	A radical slit	Only on edges of slit—no other	Loosened
38	only fringes left	Large	Loosened
40	Whole center out	Large	Loosened
41	Off on ¾ circumf.	Large	None
53	Only fringes left	Medium	Slightly loosened
54	Whole center out	Medium	Slightly loosened
55	⅔ area out	Medium	Slightly loosened
56	A wide fringe left	Large	None
57	Only a fringe	Very large	Badly loosened
58	Only a fringe	Medium	Slightly loosened
59	Only a fringe	Medium	Badly loosened
60	Only a fringe	Medium	Badly loosened
61	Completely gone	Small	Slightly loosened

TABLE IV.
Record of the "Protected" Ears.
(The left ear in each case; for the shooting arrangement, see the footnote to Table II.)

Preventive device being tested.	Guinea pig No.	Smallest diameter of the glass tube in mm.	Condition of wax packing after shooting.	Condition of tympanic membrane at autopsy.	Amount of coagulate in and on mucous membrane of the middle ear.	Evidence indicating edema of the mucous membrane.
Scientific Ear Drum Protector "Tommy"	26	2.5	OK	Intact	None	.....
	56	3.5	OK	Intact	None	None
	32	2.5	?	Intact	None	None
	25	2.5	Bit of wax in tube	Intact	One area	.....
Mallock-Armstrong Ear Defender	21	2.5	OK	Intact	Two areas	.....
	53	3.5	OK	Intact	None	None
	22	2.5	Loosened	Intact	None	.....
Wax Cone of the Italian navy type	33	3.5	OK	Intact	None	None
	34	3.5	OK	Intact	Very small	None
	57	3.5	OK	Intact	Slight	None
Cotton saturated with vaseline well worked in	60	3.5	OK	Intact	None	None
	61	3.5	OK	Intact radial streak coagulate	Along ¼ tympanic ring	Very slightly loosened
	40	2.0[1]	OK	Intact	None	None
	41	2.0[1]	Slightly loosened	Intact	None	None
Cotton saturated with glycerin-air was marked out as much as possible[2]	55	3.5	OK	Intact	Streak about tympanis ring. Two other areas	Slightly loosened
	59	3.5	OK	Intact	¾ of circumf. tympanic ring	None
	37	2.0[1]	OK	Intact	None	None
Dry cotton-packed firmly	35	3.5	OK	Hole about clot on outer surface	One area	Loosened

Preventive device being tested.	Guinea pig No.	Smallest diameter of the glass tube in mm.	Condition of wax packing after shooting.	Condition of tympanic membrane at autopsy.	Amount of coagulate in and on mucous membrane of the middle ear.	Evidence indicating edema of the mucous membrane.
Dry cotton packed firmly	58	3.5	OK	Radial slit	Edges of slit-medium elsewhere	Badly loosened
	36	2.0[1]	OK	Intact	Along 1/8 tympanic ring one other large area	.....
Elliott Perfect Ear Protector[3]	23	2.5	OK	Off on 2/3 circumf.	None	Loosened
	28	3.5	OK	Intact	Along 1/2 tympanic ring	.....
	54	3.5	OK	intact	Along all tympanic ring	Slightly loosened
Device submitted to the Council by Dr. J. G. Wilson and Prof. A. Michelson.	27	2.5	OK	intact	Along 2/3 ring other small areas	.....
			OK	intact	3 streaks along parts of rings several small areas	.....
	31	3.5	OK	Hole about 3 mm. in diameter	Several small areas one large	.....

1. Controls indicate that the 2 mm. tube is too small, so the results with it must be discounted.
2. A fourth animal was found at autopsy to have a thickened tympanic membrane due to an old infection, and so has not been included.
3. Two more animals were tried with this device; with both the wax packing was not in good condition when examined after the shooting, and accordingly the results are not included. Both showed positive injuries.

BACTERIOLOGICAL AND CLINICAL ASPECTS OF INFECTION OF THE ACCESSORY SINUSES OF THE NOSE.*

DR. J. W. BABCOCK, New York City.

This investigation was undertaken at the suggestion of Dr. C. G. Coakley, not as an inquiry into a previously unexplored province, but in an attempt to correlate the bacteriological findings with the clinical types of sinusitis, and if possible give aid in making an early prognosis, and in selecting an appropriate form of treatment.

The material used consisted of the muco-pus obtained from one hundred cases, with infected sinus or sinuses treated in the private practice of Dr. Coakley. The cases are consecutive except for the omission of a few cases seen in consultation and not afterwards, rendering it impossible to get the desired data in respect to their subsequent history. The bacteriological work was done in the laboratories of the Department of Bacteriology, College of Physicians and Surgeons, Columbia University, through the courtesy of Professor Hans Zinsser and Dr. A. K. Balls, for whose valuable suggestions and aid, grateful acknowledgment is hereby made. Acknowledgment is also made of the kindness of the Rockefeller Institute in furnishing immune pneumococcus serum.

In the majority of cases, the secretion was caught in a sterile test tube as it came from the nose while the sinus was being irrigated with a sterile solution of salt and bicarbonate of soda, the nose in all cases being previously irrigated. In a few cases the secretion could be directly aspirated and collected under strictly sterile conditions. Ethmoid cultures were made directly. The material was planted on the day of collection on bouillon, glucose-agar and blood-agar, after first attempting to overcome any possible contamination of the surface of the masses of muco-pus by several washings in sterile saline or, as was done in the later cases, by streaking it over an agar plate and taking the last of the streak for culture. Some changes were made in the routine as the work advanced. Bouillon was discarded as a primary culture medium, and anaerobic cultures were made in those cases in which a foul odor was present.

Smears of the muco-pus were made as a routine and stained with Hastings' stain, to give an idea of the cytology of the secre-

*Read before the New York Academy, Section on Laryngology, and Rhinology, Jan. 28, 1918.

tion as well as to determine the presence of bacteria. The bacterial findings in the smears very closely agreed with the subsequent findings on culture, except that a fairly large proportion of cases showing no bacteria on smears grew various organisms. Only two out of five cases in which no growth occurred, showed bacteria on smear. In one case these were long paired cocci, undoubtedly pneumococci, and in other a bacillus which was not identified.

The cytological findings showed nothing of great interest except to roughly confirm the findings of Darling,[1] who found that the presence of an excess of lymphocytes, especially when coupled with the presence of streptococci, renders the chance of cure without a radical operation distinctly less. One interesting finding was the occasional presence of a large proportion of eosinophiles, without any uniform clinical or bacterial relationship.

In considering the actual bacterial findings in the material from the sinuses, we must admit the possibility, in the majority of cases, of contamination in the specimen's passage through the nose. All the precautions thought possible were taken, but there is still some room for question. Results, however, seem to show little, if any contamination, as 69 out of 119 cultures showed only one organism or no growth. The normal nose is not usually sterile, but such organisms as streptococci and pneumococci are rarely found, according to Park and Wright.[2] The accessory sinuses are, on the contrary, found sterile in health, according to Skillern[3] and Turner and Lewis.[4] With a thorough washing of the nose as a preliminary to obtaining the cultures, the chance of contamination seems small.

The cases were rather arbitrarily divided into acute and chronic classes, depending upon the duration of the attack. Attacks lasting one month or less are called acute, and the others chronic. It is often difficult to learn the exact duration of infection in a sinus. In this series, two cases were classed as acute, although lasting five weeks, if one may judge from the history given. This was done because of their behavior under treatment, four days in case 66, and seventeen days in case 78 of irrigation of the antrum involved sufficing to clear them up. In the chronic cases especially, the duration of the attack is difficult to determine, "years" being a favorite term given. Such indefinite periods have been omitted in computing the average duration of attacks.

Of the one hundred cases, fifty-three were acute, forty having one, twelve having two and one having three sinuses involved.

These consist of involvement of the right antrum 28 the left antrum 30, the right forntal 2, the left frontal 2, the right sphenoid 3, the left sphenoid 1 and the right ethmoid 1 times, making sixty-seven cavities involved. This does not include ethmoidal involvement occurring with antra. The following combinations occurred: Right and left antrum 9; right antrum and right sphenoid 1; left antrum and left frontal 1; right antrum and right frontal 1; and the right and left antrum and left sphenoid 1. Single cavities were involved as follows: Left antrum 19, right antrum 16, left frontal 1, right frontal 1, right sphenoid 2 and right ethmoid 1 times.

Forty-seven cases were classed as chronic, thirty-four having 1, six having 2, two having 3, three having 4 and two having 5 sinuses involved. The right antrum was involved in 21 cases, the left antrum in 30, the right frontal in 5, the left frontal in 4, the right sphenoid in 3, the left sphenoid in 7 and the right ethmoid in 2 cases. This list also omits some ethmoiditis associated with maxillary sinusitis. Single sinuses were involved as follows: Right antrum 10, left antrum 19, right frontal 2, and left sphenoid 3 times. Combination of two sinuses occurred as follows: Right and left antrum 4, right ethmoid and right sphenoid 1, and right antrum and right ethmoid 1 times. Combinations of three were chronic right and left sphenoids with an acute left antrum once, right and left antrum with left frontal once. Combinations of four were right and left antrum with right ethmoid and sphenoid once; right and left antrum with right and left frontal once, and right antrum with left antrum, frontal and sphenoid once. The following combination of five occurred twice: Right and left antrum, right and left frontal and left sphenoid.

The sexes occurred as follows: Male, 28 acute and 23 chronic cases. Female, 25 acute and 24 chronic cases. No especial susceptibility of either sex is apparent.

The ages of the patients were arranged in decades. In the acute cases one case occurred in the first decade, three in the second, twelve in the third, nineteen in the fourth, eleven in the fifth, five in the sixth, and two in the seventh. The youngest patient was five and the oldest sixty-five years. The average age was 36 years. In the chronic cases, three occurred in the second decade, nine in the third, eleven in the fourth, eighteen in the fifth, five in the sixth and one in the seventh. The youngest patient was 13 and the oldest 60 years. The average age was 38 years. The difference is so slight that no conclusion seems warranted.

In the acute cases the following bacteria have been found:

PNEUMOCCOCCUS		
Type not determined	5	
Group II	9	
Group III	3	
Group IV	15	Total 32
STREPTOCOCCUS		
Hemolytic	3	
Non-hemolytic	2	Total 5
STAPHYLOCOCCUS		
Aureus	13	
Albus	17	Total 30
B. Influenza	4	
M. Catarrhalis	2	
A Diphtheroid Bacillus	2	
B. Coli Communis	2	
B. Fecalis Alkaligenes	3	
B. Aureus	1	
B. Proteus	3	
B. Subtilis	1	
No growth	4	

This makes eleven different organisms occurring eighty-five times.

The chronic cases showed:

PNEUMOCOCCUS		
Type not determined	1	
Group II	1	
Group IV	4	Total 6
STREPTOCOCCUS		
Hemolytic	15	
Non-hemolytic	4	Total 19
STAPHYLOCOCCUS		
Aureus	18	
Albus	20	Total 38
B. Influenza	2	
B. Mucous Capsulatus	5	
A Diphtheroid Bacillus	4	
B. Coli Communis	2	
B. Fecalis Alkaligenes	3	
B. Aureus	3	
B. Proteus	1	
M. Tetragenus	3	
B. Subtilis	1	
No growth	1	

This makes thirteen different organisms occurring ninety times Pure growth of one organism was found as follows:

Acute Cases	
Pneumococcus	24
Streptococcus	2
Staphylococcus	10
B. Aureus	1
Diphtheroid Bacillus	1
M. Catarrhalis	1
Chronic Cases	
Pneumococcus	3
Streptococcus	4
Staphylococcus	15
B. Mucocus Capsulatus	1
Dipheroid Bacillus	1
M. Tetragenus	1

The combinations are so varied and their relation to clinical findings so indefinite, with one exception which will be referred to later, that a detailed list seems unwarranted. Two of the organisms found, namely B. aureus and B. subtilis seem harmless saprophytes and may well be only contaminations, but were found only six times in all. B coli communis, B. fecalis alkaligenes and B. proteus seem of doubtful pathogenicity in the sinuses, and never occurred alone, so can be largely discounted as possible contaminants. The addition of a staphylococcus albus to another organism seems, however, to definitely prolong the time required for treatment in the chronic cases as will be shown. No great difference in the types of pneumococci was noted in the duration of the attacks except that Case 100 was treated longer than was usually necessary for the other types—(27 days), but Case 96, with the same Type III infection only required 4 and 6 days for her antra to clear up. The secretion was similar in both cases of a rather clear mucoid character, and Case 100 looked, from the character of the washings, as if he should clear up long before he did so.

In view of these facts, it seems advisable, in attempting to correlate the bacteriological and clinical findings, to subdivide the acute cases somewhat after the manner of Turner and Lewis[4] into pneumococcus, staphylococcus, streptococcus and miscellaneous groups. The chronic cases seem to warrant the above groups and an additional one in which B. mucosus capsulatus predominates, as that organism was only found in chronic cases.

The relationship between the bacterial groups, the duration of the disease, the time and kind of treatment and the results are as follows:

ACUTE CASES.

Pneumococcus Group 32

Time of attack, 5 to 35 days; average 11 days.

Time treated, 3 to 27 days; average 8¼ days.

All of the cases were cured by lavage of the sinuses involved and no operations for improving drainage were required.

Streptococcus Group 5

Time of attack, 5 to 24 days; average 14 days.

Time treated, 3 to 15 days; average 9 days.

Four cases were cured (80%) and the remaining case (No. 92) was much improved and left our hands to be treated in his home city. No operations were required.

Staphylococcus Group 22

Time of attack, 2 to 30 days; average 19 days.

Time treated, 2 to 19 days; average 9 days.

All the cases were cured. An operation, external incision and partial exenteration of right ethmoid was performed on Case 76, a child of 5 years, who had an extremely violent infection with orbital cellulitis.

Miscellaneous Group 3

(B. aureus, a diphtheroid bacillus and M. catarrhalis)

Time of attack, 9 to 35 days; average 18 days.

Time treated, 4 to 11 days; average 8 days.

All of the cases were cured and no operation required.

"No Growth" Group 4

Time of attack, 10 to 30 days; average 19 days.

Time treated, 6 to 12 days; average 9 days.

All of the cases were cured. No operations required.

The average time treated of all acute cases, associated with whatever organism is practically the same. The time of attack is somewhat shorter in the Streptococcus and Pneumococcus Groups.

CHRONIC CASES.

Pneumococcus Group 6

Time of attack, 2 mos. to 2 years; average 7½ months.

Time treated, 6 days to 6 months; average 55 days.

Three were associated with staphylococcus albus and were treated on an average of 41 days, one being cured and two improved. Altogether three were cured (50%) and three improved. Two (33⅓%) operations were performed, and polyps were present in one case (41) showing pneumococcus II, staphylococcus albus and micrococcus catarrhalis.

Streptococcus Group 19

Time of attack, 1 month to 13 years; average 43 months.

Time treated, 4 days to 5 months; average 48 days.

Seven cases were associated with staphylococcus albus and were treated on an average of 71 days. Sixteen were cured (84%) and three were improved, only one of these last being associated with staphylococcus albus. Polyps were present in one case (43), showing a non-hemolytic streptococcus and B. mucosus capsulatus. Eight (44%) operations were performed, by which seven were cured and one improved (Case 94), of five years' duration and treated three months.

Staphylococcus Group 21

Time of attack "months" to 20 years; average 11 years.

Time treated, 14 days to 4 years; average 171 days.

Thirteen cases (62%) were cured and eight improved. Nine (40%) operations were performed with five cures and four improvements (Cases 1-10-36 and 86).

B. Mucosus Capsulatus Group 3

Time of attacks "months" to 20 years; average 11 years.

Time treated, 13 to 98 days; average 43 days.

Two cases (66⅔%) were cured and one case (40) showing pure B. mucosus capsulatus, improved. No operations were performed.

Miscellaneous Group 3

(B. Influenza, B. fecalis alkaligenes and B. aureus together, a diphtheroid bacillus and M. tetragenus.)

Time of attack, 6 weeks to "years;" average 15 weeks.

Time treated, 3 days to 26 days; average 12 days.

All three cases were cured and no operations required.

"No Growth" Group 1

Time of attack, 1 year.

Time treated, 3 days.

Cured without operation.

The type of treatment was daily lavage through the natural orifice of the sinus involved, or in case of an antrum, through a puncture in the inferior meatus. An "operation" means a large opening between the sinus involved and the nasal cavity, except in the one instance of Case 76 where an external incision was made for acute ethmoiditis with orbital cellulitis.

Note was made of the relation between the infecting organisms of different sinuses involved in the same case. In the acute cases eight were investigated, of which six were approximately the same and two differed somewhat. They were as follows:

Pneumococcus IV—in right and left antra, three times.

Pneumococcus III—in right and left antra.

Pneumococcus IV—in right and left antra and left sphenoid with the addition of staphylococcus aureus and B. subtilis in the right antrum and right sphenoid, with the addition of B. coli communis in the right antrum.

Pneumococcus II, staphylococcus aureus and M. catarrhalis in the right antrum with staphylococcus aureus and B. proteus in the left antrum.

Micrococcus catarrhalis in the left antrum with "No Growth" in the right antrum.

In the chronic cases, 5 cases were investigated, 4 seeming to agree and one to disagree, as follows:

Streptococcus hemolytic and staphylococus albus in right antrum and frontal.

Streptococcus hemolytic and staphylococcus aureus in left and right antra with the addition of a diphtheroid bacillus in the right antrum.

Staphylococcus albus in right antrum and ethmoid with addition of M. catarrhalis in the antrum.

Staphylococcus aureus in right and left antra and left frontal with addition of B. mucosus capsulatus in the left frontal.

Pneumococcus and staphylococcus albus in the left antrum with non-homolytic streptococcus and M. catarrhalis in the right antrum.

Of agreement between present cultures and those of former attacks in the same sinus, little could be found on account of the dearth of cultures of former attacks. Four cases were, however, investigated, one of which agreed and three disagreed as follows:

Right antrum—pneumococcus IV—in 1917—and pneumococcus in 1912.

Right antrum—streptococcus hemolyticus and staphylococcus albus in 1917, and a pneumococcus earlier in 1917, and by a different laboratory.

Left antrum—staphylococcus albus, M. catarrhalis and B. fecalis alkaligenes in 1917 and streptococcus in 1913.

Righ antrum—pneumococcus II—in 1917 and staphylococcus albus and B. proteus eight months earlier in 1917.

Thirty-three other cases had had one or more previous attacks which were not cultured.

The pathway of infection of the involved sinuses was investigated and the findings do not agree with those of Turner and Lewis[5] who thought one-third of the cases of antrum infection were of dental origin. Of 109 infected antra only eight of (7½%) seemed to have any connection at all with infected tooth roots, and in none of them was any connection between the antrum and the

infected area established on removal of the tooth. However, it is admitted that the infection could pass from the tooth to the antrum without a gross connection.

The organisms found were staphylococcus 6 times, non-homolytic streptococcus, B. coli communis and M. tetragenus.

Several special antiseptics were used. Chlorazene 1-1000 and 1-100 (Case 47) and on two cases, not in this series, dichloramine T-2% (Cases 36 and 94) and ethyl-hydro-cuprein 1-1000 (Cases 61 and 100 each showing pneumococci) without any appreciable benefit being noted. In fact, the stronger solution of Chlorazene and in one case the dichloramine T, proved decidedly irritating. One of the commercial preparations of B. lactis bulgaricus was used on Case 1 for five successive days without any benefit being noted and a culture taken after a few days' interval did not show the presence of the therapeutic organism.

Attempts to determine the cause of the foul odor present in some cases reached no satisfactory conclusion. Nothing was obtained from anaerobic cultures which did not also grow aerobically. However, in one case a diphtheroid bacillus had a foul odor when grown without air, but not under aerobic conditions.

No other odoriferous bacteria were observed except those in the colon group. Turner and Lewis[5] found a foetor due to the colon group and to some anaerobes, especially those connected with tooth decay. In this series no such organisms were found.

A few cases showed special points of interest. Case 7 showed in her left antrum a staphylococcus aureus, but on a culture made some time previously by Dr. R. A. Cooke, a streptococcus was also found and streptococcus vaccine given.

It is interesting to note that the streptococcus was not found at the later culture, although there had been no clinical improvement at that time.

Case 24, a man of 62 years, had marked exophthalmos and papilitis; very slight involvement of his right antrum and sphenoid were found, showing staphylococcus aureus. These cleared up promptly and were not considered related to his eye condition which was ascribed to an intra-cranial lesion. However, a fairly prompt recovery ensued and he reported himself well a few weeks ago, over six months after his attack.

One case, Mr. W. C. J., not in this series, as he was seen but once in consultation to see if a chronic sinusitis could be the cause of an asthma of 2 years' duration, showed Aspergillus Niger. Clinically he did not resemble the cases of Aspergillosis of the sinuses as described by Tilley.[6]

It may be of some interest that eight consecutive, acute cases seen in the little epidemic of colds associated with the recent severely cold weather showed pneumococcus infection.

It is difficult to draw definite conclusions from the above array of facts. One fact, however, stands out prominently and that is the pneumococcus is pre-eminently associated with acute attacks and the streptococcus with chronic sinusitis, staphylococci being well represented in both groups. Also it is to be noted that acute cases are more often associated with a single organism.

In acute cases, obviously, the prognosis is better and the treatment simpler and less prolonged, as has long been known. Very little difference in the length and kind of treatment and results to be expected, in the different groups of acute cases, has been found. Some difference is found in chronic cases, however, and not entirely in accord with the conclusion of Turner and Lewis,[5] who consider the presence of a streptococcus of bad prognostic significance. In this series the staphylococcus group has the longest average duration treated and a low percentage (62) of cures with a high percentage of operations required (40%).

We regret having no more definite conclusions to offer than to say that as yet the bacteriological findings have not proved any great aid in either prognosis nor in determining the kind of treatment in acute cases, but are of some value in chronic cases. However, they have proved of great interest to those of us who have been doing this work, and seem to promise more on further investigation.

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53 W. 56th Street.

REPORT OF A CASE OF CHRONIC MIDDLE EAR SUPPURATION, TREATED BY THE CARREL-DAKIN METHOD, FOLLOWING A RADICAL OPERATION.*

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The patient who is good enough to come before you for examination has had, otologically speaking, a checkered career. A brief synopsis of his history as a whole may therefore be of interest to you.

I first saw him in consultation with Dr. (now Major) Bruce Phillips, on June 5, 1907. He had suffered from a chronic middle-ear suppuration of the left ear since early childhood. Two days previous to my first call he had experienced severe left ear ache, for which simple remedies were prescribed. On the day that I was called in, Dr. Phillips found him obviously seriously ill, with fever, severe occipital pain and frequent vomiting.

He was vomiting when I arrived. This was at 2 P. M. Temperature 103; pulse 120. Pupils somewhat contracted, but reacted to light. Mental condition dull,—i.e., questions put in ordinary conversational tones were answered without clear understanding or not at all. Examination of left ear showed practical destruction of membrane tensa and some tympanic granulations.

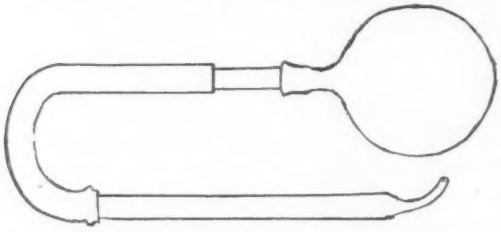
Two hours later, when the patient was removed to the Manhattan Eye and Ear Hospital, he was delirious and practically unconscious, —i. e., it was no longer possible to communicate with him. Temperature 102; pulse 125. Pupils contracted to pin points. Marked rigidity of the neck. Consultations with Dr. Van Fleet, Dr. Whiting and Dr. Bruce Phillips confirmed a diagnosis of otitic meningitis and immediate operation was decided on.

Nature of the operation. Removal of a thick mastoid cortex revealed a bone of sclerotic type. Antrum small, deep-seated and containing thick pus. No perforation of tegmen antri found. A hasty mastoidectomy was performed and the mastoid roof completely removed. From this starting point the temporo-sphenoidal lobe was uncovered by removal of the squama over an area extending about three inches, i. e., from before backwards. The dura thus exposed showed some engorgement of surface vessels and bulged to an extent indicating increased cerebral pressure.

*Notes in reference to a patient presented before the Otological Section of the New York Academy of Medcine on March 8, 1918.

The dura was now opened by three parallel vertical incisions about an inch apart, through the posterior of which a grooved director was carried directly inward and backward into the brain substance to a depth of about 2¾ inches, presumably opening the lateral ventricle, since a very copious flow of cerebral fluid followed. Loose sterile gauze was placed against the dural incisions, and a large protective dressing applied.

Results. On the following morning the temperature was 98.6; pulse 78. The patient was quite rational, but had no memory of the events of the previous day. Toward evening there was a rise of temperature and the patient became delirious. The following morning found him again rational, and from this time progress toward recovery was unbroken. During the two weeks immediately following the operation there was a continuous flow of cerebral fluid,



Rough pen and ink diagram of apparatus for introducing chlorazine paste into recesses of radical wound.

at first very copious and quickly saturating the dressing, but gradually diminishing in amount. In three to four weeks it seemed evident that recovery was assured.*

It was about this time that I suggested the advisability of a radical operation for the elimination of the tympanic focus of infection which had caused so much trouble. His family, however, were not unnaturally averse to subjecting him at this time to further operative strain or risks. A simple plastic operation was therefore done to cover and protect the exposed dural surface. The patient made a perfect recovery with the exception that the original suppurative lesion of the middle ear had not been eliminated.

From this time I did not see the patient professionally until the 20th of last November, when he gave the following history: During the past ten years he had been in excellent health. There had been very slight intermittent discharge from the left ear. At inter-

*The above condensed notes were taken from a report of this case published in the Archives of Otology, Vol. XXXVII, No. 2, 1908.

vals a small opening behind the ear would develop, which was painless, gave exit to slight discharge, usually healed fairly quickly under a protective dressing, and to which he attached no serious significance. Within the past few days, however, the sinus had reopened with appreciable swelling and tenderness behind the ear.

Examination showed a typical post-auricular swelling, but with a small opening at a point approximately on a level with the antrum roof. Owing to the past history I was naturally unwilling to make free use of a probe, but a small curette brought away a dark colored, ill-smelling mixture of fetid pus and detritus. A radical operation was advised and accepted.

Operation, November 30. Reopening of the old mastoid wound showed the antrum filled with exceeding foul detritus, and there were evidences in various directions of progressive bone necrosis. The sigmoid sinus was uncovered over a considerable area and the dura above the mastoid was covered with granulations. The tympanic vault was filled with masses of granulations and the aditus was enlarged by extensive necrosis of the inner, or tympanic, end of the postero-superior canal wall. The bone operation was completed in the usual way. A Körner flap was employed for the canal, and the posterior wound closed.

Post-operative treatment. On the table the wound cavity was flushed with Dakin's solution. A short gauze wick, saturated with the same fluid, was placed loosely in the cavity and a gauze dressing applied. On the following morning this dressing was removed and the following routine instituted: The nurse was instructed to fill the wound cavity every hour during the day with Dakin's solution, the patient lying for 20 minutes thereafter with the sound ear buried in the pillow, after which the fluid was allowed to run out, and the ear protected with sterile absorbent cotton. At night,—i.e., at the time of the last Dakin bath,—the wound cavity was dried, then filled with chlorazine paste and a protective gauze dressing applied. This treatment was faithfully and punctiliously carried out during three successive days.

To give this painstaking method a logical basis, one must remember that the theory of the Carrel treatment is based upon the hypothesis that the frequent flushing with Dakin's solution of an open wound, even though it be highly infected, will in a comparatively short time produce thorough sterilization; that chorazine paste, though not sufficiently bactericidal to insure sterilization of an infected wound, may be relied upon to prevent re-infection of a wound

which has been thoroughly disinfected. Carrel insists on laboratory evidence by smears and cultures from wound scrapings as proof of sterilization; he also implies, however, that a clinical evidence of such sterilization may be found in the complete absence of exuberant granulations.

After the third day of the routine above outlined, I assumed that the wound was surgically clean, and modified the treatment as follows: Once daily the wound cavity was thoroughly irrigated with Dakin's solution. The cavity, having been dried, was then filled with chlorazine paste and further protected by a pad of sterile cotton placed in the concha and held in position by collodion. This routine was practiced daily during the first three weeks and thereafter on alternate days.

Method of applying chlorazine paste. The use of chlorazine paste in a radical mastoid cavity requires some special appliance. It cannot very well be introduced by means of a spatula for the reason that air confined and compressed in certain recesses prevents contact. There are two parts of a radical wound cavity which call for and will repay particular care in introducing the paste, i.e., (1) the comparatively inaccessible posterior extremity of the excavation; and (2) the very easily reached region of the eustachian orifice. The accompanying figure shows a very simple contrivance which has proved convenient and satisfactory. The rather long pipette with curved extremity, or beak, is boiled with the dressing instruments and then filled from a glass syringe containing the paste. When the rubber tube and bulb are attached it is ready for use. With this it is easy to carry the paste first to the posterior recesses of the cavity and then, after inspection by reflected light, directly into the depression of the eustachian orifice. The rest of the cavity is easily filled and takes care of itself.

Dakin's fluid is more or less irritating to the skin. It is, therefore, necessary at times to protect the skin about the wound with some bland ointment. A mixture in equal parts of zinc oxide ointment and vaseline gives satisfactory results about the ear.

Notes on tissue changes observed. In the first place the bone cavity had the appearance of being surgically clean from the day of operation. With the exception of one point to which I shall refer later, there never were any granulations as usually understood. A week after the operation a thin membrane was seen on parts of the inner tympanic wall. This spread with great rapidity backward and upward toward the antrum and roof, and forward toward the

eustachian orifice, and these parts were soon—i.e., within three weeks,—apparently covered. Part of the floor and the ridge representing the remains of the posterior wall of the bany canal, on the other hand, were quite bare and here epidermization progressed very slowly.

About this time the patient and I were somewhat concerned by the breaking down of one point in the suture line behind the ear,—the site of the old sinus. This, I am sure, was due to my neglect to thoroughly curette this tract at the time of the original operation. It was an easy matter under cocaine to curette this sinus from the outside, and when this was done, I had a round opening into which an ordinary lead pencil could have been introduced. It enabled me, however, to inspect the posterior part of the wound, which apparently, except for the opening itself, was completely covered with healthy skin. The paste introduced into the back of the wound cavity now entered and filled this sinus. The closure of the sinus under this treatment was rapid.

Returning briefly to the tympanic cavity, this at the end of 4 or 5 weeks, was everywhere covered with skin except over one small area already referred to on the ridge representing the remains of the posterior canal wall, which remained bare. Hoping to stimulate epidermization, I curetted the skin edges surrounding this area, with result that it became covered by granulations but not by skin. After some days I removed these granulations with a curette. A week later I omitted the paste and substituted a dry gauze wick. In two days it was healed. I am inclined to believe that the post-operative period could have been reduced, perhaps by one-third, if the dry or gauze wick treatment had been substituted earlier.

Epitome. In actual duration of the post-operative treatment this method did not in this case result in curtailment. He was discharged as cured exactly two months after his operation. Chlorazine paste seems greatly to promote epidermization in certain parts and in others to retard it. We may count as directly attributable to this method the following advantages: (1) the bone cavity from start to finish seemed surgically clean; (2) there were no masses of granulations to contend with; (3) epidermization over essential points—e.g., the posterior recess and the eustachian orifice—was particularly rapid; (4) the treatment is practically without discomfort to the patient.

Note:—The use of a stationary rubber tube, removed only once in 24 hours, which is in routine use in military and hospital prac-

tice, does not seem to me either called for or advisable in the post-operative treatment of a radical mastoid cavity. Its use in deep-seated and infected wounds of the muscles or long bones is an obvious necessity for the reason that in no other way could the periodic flushing of the infected area be accomplished without the frequent disturbance and mechanical injury of the parts, which this treatment particularly aims to avoid. The radical mastoid wound, on the other hand, provides a natural cup, the periodic filling of which with Dakin's fluid is mechanically simpler, fulfils every theoretic requirement of the Carrel method, and obviates both the discomfort and the mechanical injury or irritation which the smallest and most carefully adjusted rubber tube might cause in this region.

Essentials to the success of this method are (a) fresh and accurately prepared Dakin's solution; (b) strict observance of the laws of asepsis; and (c) regular and careful attention to the prescribed details.

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Relation of Focal Infection to Skin Diseases. E. S. LAIN, *Jour. Oklahoma State Med. Assn.*, Jan., 1918.

In a large number of cases of psoriasis an infected tonsil or an apical abscess was discovered in about 90 per cent of the cases. In the other 10 per cent there may have been a focus of infection in some other locality. Erythema multiforme has been, in the author's investigations, almost constantly associated with apical abscesses or diseased tonsils. Of the few cases, during the past year, of lupus erythematosus that came under Dr. Lain's observation most of them were associated with diseased tonsils. Two diseases in his studies have from the beginning been constantly associated with a focal infection, namely, lichen planus and herpes zoster. He has examined quite a number of these conditions during the past two and one-half years and in every case of lichnen planus he was able to discover one or more apical abscesses. In his studies of herpes zoster approximately 100 per cent have given evidence of focal infection. Of the dermatological lesions unquestionably due to focal infection he names herpes zoster, lichen planus, erythema nodosum, with the probable addition of erythema multiforme and lupus erythematosus. ED.

A CONSIDERATION OF ASTHMA FROM A WIDER ASPECT.

Dr. Harry L. Pollock, Chicago.

It is a well known fact, that in any disease of the human economy, in which the etiology is not clearly established or is unknown, more papers are presented than on one which is well understood, each author giving his deductions and conclusions. This is especially true of bronchial asthma, a disease which affects a large number of individuals and which is classed as one of the commoner diseases.

In reviewing the literature on asthma one is amazed by the vast amount that has been written. Dr. Orville H. Brown[1] in his monograph "Asthma," gives 993 references to articles previously written, which does not nearly cover the literature on this subject, as I have satisfied myself by the perusal of the literature.

That so much has been written is perhaps due to the fact that up to the present time, no definite etiology has been established and consequently each individual has the prerogative of theorizing and bringing forth arguments to sustain his hypothesis.

My sole aim in presenting this subject is to attempt to gather the most salient points in this voluminous chaotic mass of words and present them in a tangible form, so that we may have some rational form of treatment to alleviate the dreadful condition of these victims.

That the treatment of asthma belongs primarily to the rhinolaryngologist is or should be fairly well understood by all, for the main attack lies within our jurisdiction; but at times it is necessary to call the internist to work conjointly with us.

That the severe acute attack of asthma is caused by a spasm of the musculature of the smaller bronchi, is, I believe accepted by all. This fact and this alone is the only one in which we all agree. The explanations of how this etiological factor causes this phenomenon are as varied as are the writers on the subject. For the past several years we (Dr. J. C. Beck and myself) have presented several papers touching upon this subject, and the more we study, and the more experience we have had in the examinations and treatment of these cases, the more convinced are we that the underlying etiological factors can be traced to a disharmony of the ductless glands. What the disturbing factors

causing this disharmony may be, is still a matter of conjecture; but we feel that we are on the right track and will evidently be able to throw some definite light upon the subject.

That the spasm of the bronchi is due to an irritation or stimulation of the vagi, is now also accepted by nearly everyone, as experimentally the same syndrome of asthma can be produced in animals by a mechanico-electrical stimulation of the vagus.

We know that many cases of asthma are associated with some pathological conditions of the upper respiratory tract such as suppurative ethmoiditis, with or without polypi formation; hyperplastic ethmoiditis or a suppuration of the antrum or sphenoid. Yet, that this pathological condition is not the underlying factor, is proved by the large number of patients who give evidence of these conditions without showing the slightest symptoms of asthma. These conditions certainly play an important role as an exciting cause, because after having these pathological conditions cleared up, all cases are benefited to a greater or less degree. I have yet failed to see one whose asthma became aggravated by surgical intervention on the sinuses.

In many of these cases treated by the general practitioner, the condition of hyperplastic ethmoiditis especially is overlooked, as it is not easily diagnosed, and consequently many patients are reported as being free from any pathology of the upper respiratory tracts.

Last year, before this Society, I gave our theory as to the cause of various diseases such as oto-sclerosis, ozena, osteomalacia, asthma, etc., and attempted by deductions and actual microscopic pathological findings, to prove that they all were dependent upon some change in the ductless glands. The exciting causes in the different diseases are varied, but the underlying etiological factor is a change somewhere in the system of the ductless glands.

At this point I desire to quote some abstracts from an article by Dr. Ernest Zueblin[2], which appeared in the *New York Medical Record*, March 3, 1917: "As the anatomical basis of bronchial asthma we admit;

1. A spasmodic contraction of the muscle fibers of the bronchi, arising by direct or indirect influences.

2. A swelling of the bronchial mucous membrane, a fluctuating turgescence. A diffuse hyperemic swelling.

3. A bronchiolitis exudativa.

4. Nervous impulses on the pneumo-gastric nerve and vasomotor influence."

Further on, the same author states that, according to more recent conception, the vagal center is influenced by impulses that depend on the secretions of the posterior part of the pituitary body. This complex organ containing many different nerve cells not only is supposed to be the receptor of all of the common sensibility, but also all co-ordinate involuntary motor impulses are thought to arise from this organ. Furthermore, the pituitary vagal center is made the terminal of sensory impulses from the nose. Such a fact has been brought out by Cyon's experiments demonstrating that the destruction of the pituitary body completely destroys the reflex sensibility of the nasal mucosa. Through the vagus efferent nerves, motor impulses are transmitted to the lungs and so a complete reflex arc is established. The predisposing cause of bronchial asthma is attributed to a hypersensitiveness in the posterior pituitary body, which may arise from the irritation of the bronchial mucous membrane by some substances contained in the inhaled air or substances arising from toxic or other products of imperfect catabolism. These substances are thought irritating for the bronchial sensory end organs of the vagus. Under normal conditions the afferent impulses from the bronchi, reaching the vagal centers evoke just sufficient efferent motor impulses as to create periodical contractions of the bronchi and secretions of mucous, to promote the activity of the ciliated epithelium and to insure elimination of the poisonous waste products. When, however, a hypersensitiveness of the vagal center exists, excessively violent stimuli are sent in the direction of the bronchial muscles and hence muscular constriction of the bronchi, swelling of the mucosa, and bronchostenosis takes place and asthma is the consequence.

This explanation, in my judgment, covers the very groundwork of the etiology of this disease, and most of the previous theories, while not complete, tend to improve more emphatically the above deductions.

It is but a short time since Vaughan first gave us his work on split protein poisoning as the causative agent of many of these heretofore unexplained phenomena. It is easy to comprehend that the poisons arising from the split proteins may act as an exciting factor in asthma by causing an irritation of the posterior lobe of the pituitary body or the bronchial sensory end organs of the vagus, thus creating the phenomena of asthma.

In the *Journal of Medical Research*, Sept. 1917, Dr. I. Chandler Walker,[3] of Boston, has presented his fifteenth study on asthma.

If we follow his work carefully we find that he, like most investigators, attempts to isolate the exciting cause, and avoids ascertaining the underlying or predisposing pathology. His studies are profound and of inestimable value and are well worth scrutinizing. He attempts desensitizing the patient to the protein, pollen, dust, etc., to which they are sensitive.

He determines the exciting cause by subjecting the patient to vaccination with various substances and ascertaining the one to which the patient reacts.

In those cases in which there is no reaction to any of the proteins, pollen or dust from animals, he tests the blood for agglutination with the staphy. pyogenes albus. If such cases show agglutination he vaccinates them with increasing doses of a stock vaccine of pyogenes albus. If it does not agglutinate, he isolates a diphtheroid baccilus from the sputum and uses a vaccine of this bacillus. Many experimenters have obtained good results in these cases of innoculations of proteins, and explain the etiology as being due to sensitization, clearly overlooking the fact that these agents are only exciting and not predisposing factors. As just explained, it is possible that these substances, pollens, proteins, etc., act upon and irritate the bronchial sensory end organs of the vagus and thus cause the efferent motor impulses which create this powerful stimulation to the bronchial muscles and mucous membrane, and produce the syndrome of asthma. Inversely, by not permitting the formation of these protein poisons or by not allowing the pollens and dust to come into contact with the bronchial mucous membrane, asthmatic attacks can be prevented.

The desensitization of these individuals against their special sensitizing agent, no doubt keeps the bronchial mucous membrane from becoming irritated and thus in turn prevents the efferent vagal stimuli from reaching the musculature of the small bronchi.

Another partially successful line of treatment is that which was first suggested by A. Ephraim in 1910[4]. This consists of endobronchial applications of medicaments directed to the mucous membrane by the use of the bronchoscope. He found that a novocaine-adrenalin solution, gave him excellent results. Various other remedies were employed, but not with the same degree of success. He observed during an attack of asthma, that the bronchial mucous membrane was red and swollen and a great deal of thick tenacious mucus was present. Immediately after this endobronchial treatment with novocain-adrenalin solution by the aid of the bronchoscope, the patient experienced more difficulty

in breathing, but this was only transitory for soon there was an expectoration of a great abundance of thin watery secretion and soon following this, great relief was obtained. Whether the relief was obtained by the application of the solution or by the mere mechanical irritation of the tube, was not clearly understood, but I am confident it was due to the former, as we have suspended patients in direct laryngoscopy and instilled a solution of cocaine and adrenalin and obtained similar results, so that the theory of direct irritation from the tube can be quite reasonably presumed to be incorrect. In many of Ephraim's cases only one treatment was necessary to obtain from 6 to 9 months' relief, in others it required 3 to 4 treatments and in a very small number, repeated applications direct to the mucous membrane failed to give any relief.

One of the latest lines of attack in asthma is that which was recently reported by Kahn and Emsinger[5] in the *Archives of Internal Medicine*, Oct. 1916. This treatment consists of injection of autogenous defibrinated blood. Their theory of the etiology is based on the supposition that the attack is due to anaphylaxis. As their deductions are very plausible I take the liberty of quoting the following:

"1. Asthma is due to a spasm of the smaller bronchi.

"2. Spasm of the bronchi is a manifestation of anaphylaxis.

"3. The anaphylactic phenomena may be explained on the basis of protein sensitization.

"4. Whether the protein gains access to the body by the nasopharynx, by the intestinal tract, by the respiratory system or by some other portal, it is probably absorbed by the blood, if so, it should be found in the blood especially just prior to or during an attack of asthma.

"5. The rational method of active immunization in anaphylaxis consists in repeated injections of the causal protein. If the previous premises are true in asthma, immunization by repeated parentenal injections of autogenous defibrinated blood obtained during an attack should be beneficial."

The process consists in withdrawing from 20-30 c.c. of blood and defibrinating same, as it is withdrawn. It is immediately reinjected subcutaneously and this process repeated weekly until about ten injections are given. They do not explain, however, why results are obtained by reinjecting this small amount of protein, while the whole circulating system contains vast quantities of the same protein.

The last case which they reported was in a woman who stated that whenever she became pregnant the attacks of asthma never occurred. In the course of their treatment she became pregnant again and naturally they refrained from further treatment and the asthma did not recur. This case is of extreme importance to me, as we know that during pregnancy there is an increase in size of the pituitary body and supposedly an increase of the secretion from the gland is thrown into the circulation. Is it not then probable that this over stimulation of the gland prevents the recurrence of the asthmatic attack? In a recent case which we treated by the defibrinated blood therapy, there was a marked exacerbation of the attack, which would not yield to adrenalin, but did to one injection of pituitrin, which acted almost as a specific. As far as the action of a hypodemic injection of adrenalin is concerned, it is entirely useless for me to state, as you well know, that usually one injection of from 10 to 15 mms. stops the attack immediately. We have found, however, that if it is given with equal amount of normal salt solution, the action is more efficacious, and of longer duration. At this point I desire to call your attention to the foolhardy practice of many physicians in placing the adrenalin and hypodermic syringe at the patient's disposal, as they very rapidly acquire the adrenalin habit. We have under observation, a patient that we found using hyperdomatically as much as one ounce per day of the 1-1000 solution. We have seen others who were injecting as high as 20 mms. every 2-3 hours. This adrenalin habit is much more difficult to combat than is the morphine habit, as the patients crave and beg for it and become desperate if it is withheld. In most cases we have found it extremely difficult to break them of their desire for adrenalin and permit us to put them on any other line of treatment.

As many patients obtain beneficial results from these various methods of treatment enumerated, it is our custom to give them the benefit of a systematic course, one after the other, until the patient has had beneficial results, or until all known methods have been exhausted.

I shall now outline our course of treatment in these cases. If the patient is first seen during an acute attack, our aim is to give him immediate relief, as that is what he seeks. A hypodermic injection of adrenalin 10 to 15 mms. is given and repeated when necessary, often at intervals of from two to three hours. Then the patient is thoroughly examined, going into the history, laboratory and physical findings. The bowels are cleaned out thoroughly

and a diet is given as free from protein as possible. The patient is kept at rest away from the noise and dust. Usually, in the course of a few days, the acute attack has subsided and then we are ready for the surgical interference of the upper respiratory tract, providing a pathological condition is found. As previously stated, most of the patients give evidence of some pathology in this region, either suppurative or nonsuppuratiye, and this must be corrected as far as possible. The ethmoids are exenterated and the other sinuses are explored, for whatever is necessary must be done thoroughly. In the meantime, the excretions and secretions are given proper attention and the above diet is adhered to. Often, during this part of the treatment, it is necessary to resort to the use of the ductless glands products, either adrenalin or pituitrin. We have the internist always associated in the treatment and management of these cases.

If the patient does not give any evidence of pathology in the upper respiratory tract, we then turn to the internist for aid. The patient is given a thorough physical examination, and if any focus of focal infection is found, such as a chronic appendicitis, a choleocystitis or what not, this is then eliminated. If nothing is found we proceed to further treatment.

The patient is then given a systematic treatment of application of cocaine and adrenalin by the bronchoscope or suspension laryngoscopy. The latter procedure is employed more often as a better view of the larynx and trachea is obtained. It is not difficult to pass the bronchoscopic tube while the patient is suspended to make application direct to the bronchi.

Since studying the reports of the treatment by re-injection of defibrinated blood, in conjunction with our internist, we treated a number of cases by this method, but our results were much different than those of the original investigators. They report that they had no immediate bad results from the injection, but in several of ours we obtained an anaphylactic reaction, resulting in profound cyanosis coming on within an hour after injection. In one of these, one dose of pituitrin cut short the attack at once. We have for the present discontinued this line of treatment.

Conclusions:—1. The underlying causative factor of asthma is undoubtedly due to a disharmony in the ductless gland system.

2. The exciting factor varies; dust, pollens, excreta from animals, split proteins, poisoning, etc., but unless there exists a disharmony in the ductless gland, asthma cannot result.

3. A systematic line of treatment such as is suggested in this paper will give a beneficial result.

4. To attempt a permanent cure, we must study our ductless gland system more thoroughly and that will come as soon as the physiological chemists will have proved such conditions by experiments. All we can do as clinicians is to examine our cases carefully and treat them on a rational, but at present, empirical basis. The reports should always be as enthusiastic as possible, yet absolutely truthful.

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Pituitrin in Tonsillar and Nasal Hemorrhage. S. Salinger, *Therapeutic Gazette*, Jan. 15, 1918.

On the basis of 87 tonsillectomies (52 under general, 35 under local anesthesia) the results with pituitrin used as a hemostatic in the presence of hemorrhage have been very gratifying. In five cases of secondary tonsillar hemorrhage the pituitrin alone was sufficient to control the bleeding. In six cases of secondary nasal hemorrhage following operation in all but one the pituitrin alone brought about a cessation of the bleeding within an average of 15 minutes after injection. In the unsuccessful case the hemorrhage came from an artery in the base of the septum which must have been sliced in its long axis, making its obliteration by packing difficult as there would be a violent hemorrhage upon removal of the pack until finally it was left in situ for five days. Where a secondary hemorrhage occurs, unless it is arterial, the pituitrin is successful and in most cases will obviate the necessity of repacking the nose. Ed.

TRICHLORACETIC ACID A SPECIFIC IN VINCENT'S ANGINA

DR. THOMAS J. GALLAHER, Denver.

In looking over the latest bibliography in regard to Vincent's angina I find a great many remedies recommended, but no mention is made of one remedy which we have used for many years with remarkable success.* I have no hesitation in pronouncing trichloracetic acid a specific in this disease.

In view of the fact that Vincent's Angina is alarmingly prevalent among the soldiers in our different cantonments, I beg to bring this remedy to the notice of the profession and of the authorities.

It should be applied pure, as follows: A small applicator, such as used by nose, throat and ear men, is wound with cotton and dipped in the pure liquid acid. The excess is removed by touching it against an ordinary blotter, and the acid is carefully applied to the entire area affected. In case of the tonsils, the acid should be carried on a thin applicator to the depth of each crypt involved, in addition to the surface application. After the parts are turned white it should be neutralized in one or two minutes by the application of a saturated solution of sodium bicarbonate. The application of the acid and its neutralization may be repeated in two or three days if necessary. The results will be dependent upon the thoroughness of the application. We have rarely found it necessary to make over two applications. An excess of acid must not be used, and none must be permitted to fall into the larynx.

*(Editor's Note—Dr. Gallaher is mistaken as to the absence of literature on the use of trichloracetic acid in Vincent's angina. Like every other remedy that has been proposed, this drug has been used with varying results. Halsted and Greene recommended it in 4 per cent solution, but even those who have used it in full strength have not met with invariable success. Reference to the use of trichloracetic acid in Vincent's angina may be found in the following articles: Dr. Frank Carroll, "Vincent's Angina," The Laryngoscope, Oct., 1917; Dr. J. M. Hester, "Vincent's Angina," American Jour. of Surgery, Feb., 1918; Dr. J. M. King, "Vincent's Angina," California State Jour. Medicine, July, 1918.—P. F.)

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

December 20, 1917.

The Treatment of Stenosis of the Larynx and Trachea Following Diphtheria. Lantern Slides and Illustrative Cases. DR. HENRY LOWNDS LYNAH.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. JOHN ROGERS said that it was a great satisfaction to hear Dr. Lynah's statement that there were now probably no cases of chronic laryngeal or tracheal stenosis following diphtheria or tracheotomy which are not curable by one or another line of treatment. Dr. Rogers said that he became interested in this subject fifteen or twenty years ago, and at that time it was almost never possible to cure these cases. He began by laryngo-fissure and attempted to remove the thickened tissue and cicatrices, but found that after incision of the tissue which caused the obstruction the cases were worse than after the original condition. Also these cases lost part of the thyroid cricoid cartilage both. It was interesting to hear Dr. Lynah say that a number of his cases did the same thing, but the cartilage returned. Since these fifteen years have passed, it has been interesting to see the final outcome of these cases. Last month he heard from one of these children who is now a young man and in the army. He began his letter by saying that he had recovered splendidly and hoped Dr. Rogers would send him to college as soon as his army life was ended. His voice had nearly recovered in spite of the loss of a large part of the cartilage, leaving him only a slight huskiness. Of three or four other patients, one has been through Vassar College and is now a teacher and using her voice constantly. These were all handled in the way that Dr. Lynah advises—by long continued dilating pressure.

Dr. Rogers said he had been much interested in the retention methods for preventing auto-extubation. The method which he had first devised was not quite so good as that now employed by Dr. Lynah and not so satisfactory. He had adopted a right angled arm fastened to the tube and inserted through a tracheae fistula. In some instances it worked pretty well; in others, not.

The outlook for these cases now is apparently extremely good and Dr. Lynah was to be congratulated on the excellence of his results. It was to be hoped that his method would become more widely understood and applied.

DR. EMIL MAYER said that the laryngologist who sees the ultimate result of these conditions rather than as they are seen in institutions almost immediately after the affection has been occasioned, can hardly appreciate the marvelous difference. Stenoses of the larynx are apparently very much more rare now than in the early days when O'Dwyer was still with us and showed us how he performed intubation; for in the more recent years they have become still less.

It is well to bear in mind, however, that diphtheria is not the only condition which produces laryngeal stenosis, it may be caused by typhoi

fever and other infectious conditions. Dr. Mayer said that his advice in the treatment of these cases might be epitomized by saying: Don't do any surgery. Remember that the larynx is extremely tolerant of intralaryngeal methods of treatment. It is remarkable how much one can stretch the interior. Frequently the ordinary uterine dilator can be used with good effect.

Dr. Mayer said he was surprised to hear Dr. Rogers speak so lightly of his wonderful right-angled apparatus, which he himself has found to be of tremendous value. One of the points which the essayist said deserved to be especially endorsed: Give your patient time to recover. In some instances it will take a long time, but the laryngologist can offer no higher service to humanity than the patient long-continued treatment of these unfortunate cases.

Dr. William P. Northrop said that for one to be the oldest living anything was a rather unenviable distinction. Dr. O'Dwyer, of course, was the pioneer of this work, both inside the hospital and outside. While he was making and perfecting these tubes, Dr. Northrup was making the autopsies. Dr. O'Dwyer put the tubes in, and he dug them out. Later the results were better. At that time Dr. Dillon Brown was the house physician; he practiced putting in the tubes in the hospital, and later on outside. The pathologist was the other. The others have passed on. In the adjoining room was an exhibit of the tubes O'Dwyer invented and perfected, and it was a notable fact that the tubes have never been modified since he died. It was five years before he would allow anyone to speak of them outside; after he had finished it and it was ready for the open market only one thing was changed. That was that the mouth gag was improved. That was the only change ever made. Dr. Brown was a clever operator and had good results. Dr. Northrup said that he had 125 intubations outside the hospital, and in the experience of all three of them he could not recall a single case of retained intubation. He had asked Dr. Lynah how he accounted for this, and he replied that they saw these cases early in private practice, and still earlier in the hospital; that seems to explain the point satisfactorily. Also in these days of anti-toxin more cases lived long enough to have after effects.

Dr. Northrup said that his experience with these cases terminated with the coming of anti-toxin, which simply swept them out of the field. Before that he had sometimes had three tubes in at once, and the watchfulness required made those busy times. O'Dwyer's first work was the insertion of the tube and making it anatomically; his second work was intubation for chronic stenosis, such as syphilitic stenosis, etc. Kelly Simpson was very good at that. He was working on the third problem at the time of his death, namely retained intubation tubes.

Dr. Northrup said that he had been much edified with Dr. Lynah's demonstration. It took him back to the old times as he was looking over the field covered. All of these conditions looked like old friends. Dr. Clinton Wagner had operated on a case in which it was impossible to get rid of the tube, and the patient recovered his voice pretty well. He operated through the wound and polypoid tissue. This was the only case he recalled in which there was extreme difficulty, and he did not know the reason for it.

Dr. H. L. Swain (New Haven) said he had come to the meeting to learn, not to express his opinions, especially in view of the demonstration made by Dr. Lynah. It was a great privilege to see and hear of such work.

Dr. Northrup had wondered why it was that they had seen so few of these cases of retained tubes in past years while Dr. Lynah sees so many now. The difference may lie in the character of its inflammation. These things come in waves. In New Haven, he himself had not seen a case of retained tube for years until within the last month when they had four cases at one time. Also they had not had any cases tracheotomized for

a long time, and then four cases came in requiring such treatment, illustrating how these cases come in groups.

The demonstration made by Dr. Lynah showed the great development of Dr. O'Dwyer's work since his time because we have had the advantage of direct endoscopy. Especially in cases of acute stenosis due to diphtheria Dr. Lynah has shown us how by suction we can remove the obstruction from the larynx and large tubes through the bronchoscopes which had been shown.

DR. DELAVAN said that the address of the evening should arouse the patriotism of every man present. It should be realized with satisfaction that the principles underlying the treatment of chronic stenosis of the larynx had been first discovered and applied in New York City and that their full development had been carried out by New York men. Horace Green had been the first to recognize the important physiological principle of the tolerance of the larynx to the presence of a foreign body. Joseph O'Dwyer, going a long step in advance, demonstrated the tolerance of the larynx to the continued presence of a foreign body. He then proceeded to perfect the art of intubation for the relief of acute laryngeal obstructions. Later he entered upon the study of the cure of chronic cicatricial stricture of the larynx and trachea. Something had already been accomplished in the successful treatment of cicatricial webs of the larynx by dilatation—or by cutting of the web followed by dilatation—by such methods as those of Sir Morell Mackenzie, Whistler, Schroetter, Gleitsmann, and others, and the operation of thyrotomy for the deeper and more diffused strictures had been successfully practiced and perfected by Clinton Wagner. By the above mentioned methods of dilatation, however, the stretching of the scar tissue was not very vigorous, and it could only be applied at intervals and for a few minutes or less at a time. O'Dwyer taught that dilatation, to be effective in the more stubborn cases, must be applied by a dilator made of hard, unyielding material, and that the stretching of the cicatricial bands must be maintained at a maximum and continuously over long periods of time. Dr. O'Dwyer died before he had perfected the execution of this idea. Subsequently, Dr. John Rogers undertook to elaborate it. His success was complete, and for certain types of cases his method has not been surpassed, permitting, as it does, continued variable dilatation of the constricted areas with a minimum of discomfort to the patients, with perfect security in the retention of the tube, and without further extended operation in cases where a tracheal cannula has already been worn. The value and effectiveness of this method is far greater than Dr. Rogers has led us to suppose, if we may judge by the number of patients he has cured and by their appreciation of his work. Much time and patience must sometimes be used in its application, but with these a far greater degree of ultimate success can often be obtained than by the use of harsher means.

Good discoveries and inventions have not seldom been lost or else set aside and forgotten. With O'Dwyer, time has only added to the world's appreciation of his genius to the value of his grand original ideas. Thus, in 1913, a tablet was placed in the diphtheria department of the Stephanie Children's Hospital in Budapest, "In honor of Dr. O'Dwyer and to commemorate the 2000th performance of intubation in that institution."

Far in advance of anything done abroad, even including the excellent work of Sargnon and Balatier, Dr. Lynah has continued the successes of O'Dwyer and Rogers until there are few cases, however difficult, which are not amenable to relief at his skilled hands. His demonstration has been so clear and graphic as to make his methods readily understood by all.

Gurdon Buck, the father of intra-laryngeal surgery; Horace Green, Clinton Wagner, John Rogers, and the lecturer of the evening were all citizens of New York and members of the Academy of Medicine—a fact of which we as fellow-members have reason to be proud.

DR. LE WALD said that he had had the privilege of helping Dr. Lynah control the treatment of some of the cases by the use of radiography, and had been much impressed with his extreme patience in caring for them. Dr. Le Wald said that the case of bronchiectasis in the *upper* lobe of the right lung had possessed especial interest for him. He had not observed a similar case, and so few successful cases of this type have been recorded that this one stands out prominently. The recovery of the voice in the case of ossification of the cartileges was remarkable. As Dr. Lynah said, the question of the thymic complication was interesting. In one instance the treatment of the thymus gland resulted in such improvement, that they were led to believe that the pressure from the large thymus was an important element in keeping up the stenosis of the larynx.

DR. LYNAH agreed with Dr. Mayer and Dr. Delavan that Dr. Rogers was entirely too modest in regard to his own valuable work in this subject. Dr. Rogers was a pioneer in the work, and when he himself was on the House Staff in the Willard Parker Hospital Dr. Rogers was experimenting with the tubes and was treating the chronic cases with successful dilatations by means of special tubes, and cured quite a number of them by his methods. The clamp which Dr. Rogers devised has never been improved upon any more than O'Dwyer's tube, which he uses every day.

Dr. Lynah said that the chief difficulty he met was with the dense scar tissue that forms in cases of laryngostomy. Dr. Jackson treats these cases by keeping them open until all the scar tissue has been absorbed, and thus he gets good breathing space and a good speaking voice. He secured some wonderful results; in one instance a girl whom he treated recovered with a good singing voice.

Dr. Lynah said that he had, however, found it better to dilate these cases and fall back on the Rogers' tubes. In his experience, the voice is usually better in cases treated by intubation rather than by laryngostomy. It is remarkable what the larynx will tolerate and how it will return to the normal if one only has patience. Sometimes one may be so discouraged as to feel that he knows nothing whatever, and yet just as he feels compelled to acknowledge a failure the work will count and the patient recovers. There are many puzzling features in these cases.

Dr. Le Wald had done all the radiographic work in these cases and had made some remarkable pictures. Dr. Lynah said that he had hoped to have the picture of the thymus case to which Dr. Le Wald had referred, but the Doctor had been too busy with his army work.

Meeting of January 23, 1918.

Bacteriological and Clinical Aspects of Infections of the Accessary Sinuses of the Nose. DR. JOSEPH W. BABCOCK.

(*Published in the present issue of* THE LARYNGSCOPE.)

DR. SIMON FLEXNER expressed his gratification at the opportunity of participating in the interesting program of the evening. Having himself been a not infrequent sufferer from affections of the upper respiratory tract he had, at somewhat irregular intervals, been interested in the subject over a long period of time, although his bacteriological studies of that region had not been carried on systematically. In the course of those studies he had consulted with Dr. Coakley who provided the suitable material. The studies were cultural and microscopic, but they were not to be entirely relied upon for decision. Th cultures were made upon media containing blood, and under ordinary and also anaerobic conditions, the latter, however, not the strictest.

The fact that attracted most attention was not the large but the small numbers of bacteria that were found. The examinations were made

at certain periods during which the infection continued, and they altogether lacked the striking quality of the paper presented by Dr. Babcock, namely the systematic and thorough study of many cases as they occur in practice, and with reference to the duration as well as the clinical conditions of each case.

Naturally, however, the interest excited by this very irregular study carried on by Dr. Flexner with the co-operation of Dr. Coakley gave to the evening's discussion personal interest that it might otherwise have lacked. In his remarks he would naturally consider the paper as a bacteriologist and not in regard to its clinical aspects.

The present views in regard to bacterial varieties differ widely from those held only a few years ago. The best illustration of the modern point of view is afforded by the pneumococcus. Most of those present doubtless remember when it was said and taught that practically every one carried pneumococci in his nasopharyngeal secretions and this condition was viewed rather hopelessly in regard to the prevention of pneumonia.

We have learned since then that the pneumococci do not form a single species, but comprise a group having certain common morphological appearances and cultural properties but differing in their immunological reactions. The several varieties or types are distinguished not as they appear under the microscope, nor as they grow in cultures, nor as they act on sugars which are fermented, but exclusively according to the way in which they group themselves when they are submitted to the tests of blood serum of animals immunized to different members of the group. That has proved a most illuminating method of study. It has been shown that the number of persons who harbor pneumococci in their secretions is not 90 per cent, but about 50 per cent, and nearly all those usually carried are of the less pathogenic variety. Hence the pneumococci which are present in the respiratory tract under ordinary circumstances do not tend to produce pathological conditions. The serious pathological states of these membranes are caused not by the pneumococci commonly present, but by pathogenic varieties which are extraneous. The latter are to be regarded as having been introduced from without and as having no normal relations to the flora of those membranes: they are as much out of place in the nasopharynx as would be the diphtheria bacillus.

Dr. Babcock has in his paper defined the immunological reactions of the pneumococci obtained in his cultures. As you will have observed, he found among them representatives of the extraneous and pathogenic types. It is perhaps still too early to draw exact conclusions as to the significance of the pathogenic pneumococci in sinus infections, but since those types are usually associated with pathological states, it is probable that they are not mere indifferent comitants of other more injurious bacteria.

Perhaps a more difficult undertaking is to deal adequately with streptococci in relation to the pathological conditions. Our knowledge of streptococci, while voluminous, is not nearly so complete and clear as in the instance of pneumococci. We possess few criteria to distingush saprophytic from parasitic varieties and the sub-varieties of the larger groups. Hence it is difficult to define the significance of streptococci

as present in sinus infections. Streptococci are very commonly present on the normal nasal mucosa, and while the haemolytic possess perhaps more significance than the non-haemolytic varieties, yet it would be a mistake to throw out all the former as of no account and merely incidentally present in the muco-purulent exudate.

Anything which contributes to the solution of the streptococcus problem, either by finding ways to distinguish the micro-organisms by specific grouping, or by obtaining more information as to their pathogenic action, would be of very great importance. Dr. Babcock in his important work made an effort to bring in group distinctions, and it it is to be hoped that he will continue that study and reach a more definite goal.

As regards the diphtheroids, a very good case would need to be made out for them in order to establish pathological significance. They are ubiquitous organisms, are present in dust, and hence could easily enter and become implanted on a preexisting pathological condition. The colon bacilli are, of course, extraneous also but they may possess pathological significance. The mere fact that they survive in competition with the normal flora of the upper respiratory tract is in itself suggestive of activity.

Dr. Flexner stated that no deduction could be hazarded regarding the staphylococci found by Dr. Babcock. Without more information regarding their pathogenicity—a fact determinable in the rabbit—judgment would need to be withheld. The white staphylococcus was, of course, a common saprophyte of the nose and throat; and the yellow staphylococcus might also be present in the mucosa without doing damage.

The value of such a paper as Dr. Babcock's, he ventured to suggest, was in direct proportion to the precision of the data submitted for analysis. It was always highly satisfactory to have presented systematic and critical bacteriological studies of closely observed clinical conditions, extending over considerable periods of time. The light which they may throw on the etiology and clinical tendency of the conditions may at any time become strong and illuminating, while fragmentary and incomplete studies are more likely to be misleading than informative. It is to be hoped, therefore, that Dr. Babcock will continue his studies along the lines pursued by him until he reaches more definite conclusions.

Dr. A. K. Ball., expressed his appreciation of and interest in Dr. Babcock's paper from both a personal and scientific point of view, as the report of his own case was included in those shown on the charts. His interest in the subject was concerned with the chemical aspects, and he had been specially interested in the reports of the various antiseptics which Dr. Babcock had employed in his work.

With regard to the use of antiseptics, the report was not very favorable, but when one considers the large number of antiseptics in use and the different properties they possess, it is not surprising that the ideal one should not be found in the first attempt. A great deal about the properties of antiseptics is being learned now, and we are especially interested in their use and application in the present war. We are beginning to realize that the bactericidal power of a substance is not the only critereon of its efficacy. A substance which is capable

of killing bacteria in great dilutions in the test tube is not necessarily effective as an antiseptic in the human body. The reason is obviously that bacteria in vitro are exposed and free to the attacks of the antiseptic used, but in the human body as an infecting agent they are usually embedded in tissue, or covered with mucous material, and thus are surrounded by organic material of one sort or another, generally protein in nature. Such protein material is often as capable of combining with the active principle of the disinfectant as is the bacterial body

With organisms so protected, the antiseptic is practically wasted on the protective layer and may never get near the organism. This principle is particularly applicable to the group of inorganic salts, silver nitrate, for example, being able to combine with proteins so readily that it will be thus rendered innocuous long before it penetrates to the interior of the mass and attacks the micro-organism.

So, in considering the value of an antiseptic, one must consider not only its bactericidal power but its penetrating power, the ability to sterilize organic material "at a distance." It must act on organisms below the surface treated. The much-maligned antiseptics of the phenol group possess that power to a greater extent than the inorganic substances. They can penetrate into the tissue and sterilize it where inorganic salts in themselves highly toxin to the "naked" bacteria cannot reach.

Another group of substances, the terpenes, to which belongs turpentine, do not kill bacteria rapidly, but they possess the power of inhibiting growth and of penetrating through organic tissue. In doing so, they are able to prevent the multiplication of the organisms and thus enable the ordinary defensive factors of the body to overcome the bacteria. This indicates another direction in which thought could be directed.

It is also interesting to speculate as to whether or not a highly present in such infections as Dr. Babcock describes would have any potent bactericidal serum prepared against the particular organism effect if used as an irrigating agent. Experiments along that line could well be undertaken. It is not difficult to obtain such sera; harm to the patient would be impossible, and theoretically at least, it should be reasonable to expect good results. Whether or not the theory would work out in practice, one cannot say.

The new thoughts concerning antiseptics are as yet mostly theoretical, but lead to certain definite lines of experimentation, and it was to be hoped that at some future time Dr. Babcock would be able to do some work along these lines.

Dr. Dwyer said that at the Manhattan Eye, Ear and Throat Hospital, they had been studying these cases for over seven years and had pretty complete records of what had been done. They had studied the question from all angles. Eighty-five normal noses had been examined, noses of patients who came in for ear treatment and in whom there was a negative history of nasal trouble. All of the organisms found by Dr. Babcock had been found in these noses and during the last three years a differentiation of the pneumococcus had been practiced. To follow the cases from start to finish, the consideration of the acute coryzas would be in order, as many went on to sinus

disease. Inn the great majority of acute coryzas, no organism could be demonstrated and in such cases this was thought to be due to the acidity of the secretion or that possibly the causal organism belonged to the filterable virus class, as suggested by Tunicliffe. The question arises then that if these organisms are found in the normal nose and if in diseased conditions we find one set of organisms on the one side and a different set on the other in the same patient, to which set are we to attribute the lesion or to any set at all. It was his opinion that bacteriologically we had still a long way to go yet before the problem was elucidated.

Dr. Dwyer would like to ask Dr. Babcock what in his opinion or in his paper he called a "cured" case. Contrary to the opinion held by some that the nasal chambers were normally free from bacteria, Dr. Dwyer had found time and again bacteria of the various kinds mentioned in normal noses and thinks that the above statement is one without any real basis in fact. Did Dr. Babcock consider the cases cured in a clinical or in a bacteriological sense?

Dr. Dwyer said he was glad that Dr. Flexner had brought up the point of staphylococcus pyogenes albus infections. A year ago at the Manhattan they had a very severe case of mastoiditis, sinus thrombosis, pyaemia and death due to this organism and since that time had been investigating the various pathogenic or likely pathogenic strains. Six such cases together with some research work on them were reported at the last meeting of the American Otological Society and a total of 22 were being reported next week at Boston by Dr. Wendell C. Phillips.

The organism had not yet been tried on rabbits, but had been tested on guinea pigs, for the dosage could be tested, and one or two cc would kill a guinea pig in five days, sometimes with septicaemia and sometimes with pyaemia. Dr. Dwyer said he would advise anyone who found albus in a case to look out for a low grade infection which might give a great deal of trouble.

In regard to treatment, Dr. Dwyer said it had always been a question with him how lavage or washing out could affect sinus disease. Dr. Haskin has a large collection of over two hundred bones showing sinus formations, and it was difficult to see how one could wash out the ethmoid or the sphenoid in most cases. Therefore, for the last few years, at the Manhattan they had given up lavage and were employing suction, and felt that they had obtained splendid results that way, with a shorter duration of the treatment. All of these conclusions were obtained from an experience of 300 to 400 cases.

Dr. Coakley said that the work done by Dr. Babcock was enormous in amount, and in addition to his other clinical work he had undertaken to work out these bacteriological findings at his suggestion. He deserved much credit for this arduous and painstaking work, which not every one was fitted for or had the patience to carry out. Dr. Coakley said that he had tried to have similar work done heretofore but it has been rather desultory. The bacteriological examinations in out-patient practice is unsatisfactory for it is impossible to follow up the clinical side of the cases, as the patients disappear. It was to be hoped that Dr. Babcock would continue this work until he gets not only a hundred but several hundred cases, when more practical deductions may be secured.

The question of antiseptics or disinfectants from the practical standpoint, has been investigated and the conclusion is that any one of these substances which he has used, if used in sufficient strength to kill the bacteria, has produced such inflammation and irritation, that the result, from the clinical standpoint, has been harmful. It has caused discomfort to the patient, and has been followed by increase in the discharge, so that he has desisted from using antiseptics and disinfectants, and employs normal salt solution with half the strength of sodium bicarbonate in all irrigation, either of the nose or accessory sinuses. This has seemed to do most good and least harm.

The suggestion made by Dr. Ball that antiseptics do not penetrate deeply into the mucous membranes, into the folds or into the tissue itself is a very good point. It is doubtless one of the reasons why we do not get more benefit from them viz., that the antiseptics act on a few organisms on the surface, and the inflammation of the membranes makes a better culture material in which the bacteria can reproduce and consequently antiseptics have a harmful instead of a beneficial result. If we can get material which is not only bactericidal but will also act upon the bacteria in the interior of the tissues without irritating the parts it will be a very great advance and will enable us to do more therapeutically with antiseptics than we can do at present.

One thing which Dr. Coakley had hoped for from this work was to get some explanation of why it is that so many patients have recurring attacks of infection of the sinuses. That is a very puzzling problem at present. If he remembered rightly, 20 per cent of the patients were cases that had more than one attack. That is a very common thing in his own practice, and it is not always in the same sinus. If it were the same sinus, say the right maxillary sinus, one might expect that there was a residue in the sinus or some condition in the mouth around a diseased tooth not recognized, by which the maxillary sinus became infected; but when a patient comes in in the spring with a right maxillary sinus infection, and comes in in the fall with the left invoved and with a different organism, the old explanation that there might be some peculiar anatomical arrangement by which they became more easily infected, a large turbinate, etc., will have to be abandoned and we will have to look further for an explanation that will be satisfactory. Some of these patients escape for a year, or may even go two or three years without an attack, but they usually recur sooner or later. Some of his case records run back ten years, and he finds that some patients have an attack nearly every year, some only once in two or three years, but a great number of acute attacks involving one or two accessory sinuses, and not always the same.

The question of the condition of the sinus when the patient is discharged as cured, is an interesting one. Dr. Coakley says that he holds that in the normal sinus there is no accumulation or retention of a mucoid or mucopurulent secretion. A normal sinus is free from secretion. The presence of an appreciable amount of muco-purulent secretion in any sinus is an indication that the sinus is or has been the seat of an acute inflammation. The easiest sinus to diagnose and treat, though not always to cure, is the maxillary sinus. Transillumina-

tion affords a pretty good indication of what is going on in most instances. One may be fooled occasionally by differences in the transillumination, but if you feel a little suspicious wash out the cavity. If the sinus is not involved the fluid will return free without any sensation of resistance on the part of retained fluid. When a patient has an acute sinus and you wash out two or three drams of thick or thin material, and the next day a little less, and transilluminate each day, as the secretion subsides you will find that the transillumination becomes more satisfactory, and finally on transillumination it is perfectly clear, and as good on one side as on the other, you can be pretty sure that there is nothing there and that the patient is practically normal. Then, if you have that patient return a few weeks later and find him apparently perfectly well, with no discharge and no interference with the transillumination, that patient can be called a cured case. Dr. Coakley said that they had not at any time examined the nasal cavity to see whether bacteria were within. He was under the impression that the interior of the nasal cavity, beyond the vestibule, say the first half inch within the nose, that beyond that point to pretty close to the naso-pharynx it was under ordinary conditions nearly or entirely free from bacteria; and that bacteria for the most part were found in the very front part of the nose.

Cobb, of Boston, had carried on an investigation which had been previously made abroad, and got the impression that the interior of the nose was nearly free from organisms; therefore, Dr. Coakley said, he could not quite see why, if one is careful in passing the instrument into the nose, he should find bacteria within the cavity; if found well within the nose it was the fault of the technique in passing the applicator, which became contaminated at the vestibule, and the organisms found did not really inhabit the interior but were carried into it from the vestibule. He could not therefore agree with Dr. Dwyer that we ought to examine the interior of the nose to determine whether the antrum is free from bacteria. He did not see how we can examine it and make sure that the antrum is free of bacteria during life, without resorting to a very large trochar passed into the antrum under aseptic precautions and pass through the trochar a platinum loop which will curl up and reach the several parts of that cavity. Very few patients would submit to that experiment in the intervals of attacks, and it would be difficult to pass such a large instrument without carrying bacteria into the antrum.

His own feeling was that when the antrum is washed out and the patient has gotten through with one of the acute attacks and the congestion has subsided, there are no bacteria in the antrum. The experiments made on the cadaver shortly after death show that in many cases the antrum is free of bacteria.

The question of treatment, whether suction or irrigation should be employed, is largely a matter of clinical experience; on the part of those who are using one or the other method. We do not claim to wash out any ethmoids; we do not attempt to wash out the ordinary acute frontal sinus, and probably could not do it if we tried. We do sometimes wash out an acute sphenoid. We have no hesitancy in attempting that, and usually succeed in passing a probe, immediately

followed by a small sized canula and washing it out. It is nearly as easy to wash out the sphenoid as to wash out the maxillary sinus.

The reason he did not approve of the use of suction was that he had not been able to get out of the cavities that were immediately involved more than a small modicum of the thick tenacious secretion found there. He had tried it, and after having produced such a high degree of suction as to cause some mucous membrane hemorrhages and even to get in the suction tip a little blood along with serous and mucoid secretion, he had then, in order to satisfy himself, introduced the canula into the antrum and washed out as much secretion as if the suction had not been applied. He could not therefore believe that suction would remove thick tenacious muco-purulent secretion from the maxillary antrum and leave it as clean as he could get it by washing out the sinus and blowing out the residue. He had tried suction often, and had ceased to use it. In some cases it does positive harm. In some cases where it has been used he has seen oedema and swelling of the mucous membrane that has not been seen in any case unless suction has been employed, and that oedma and swelling have exactly the opposite effect to that which is desired. We want to keep down the swelling in the nasal chambers instead of increasing it and so hindering drainage from the various chambers.

Dr. Abraham said that several years ago at the Polyclinic he began to investigate the bacteriology of sinus infections. He was not himself a bacteriologist, but the work was carried on by Dr. Jeffries and others, and he was impressed by the fact that the findings then obtained were practically those presented this evening. Many of the reports came in as mixed infections, and it could not be determined what was the predominating organism, so the work was discontinued.

In regard to the acute cases, he agreed with all of Dr. Coakley's contentions, that in the majority of cases simple washing out with normal saline solution is all that is necessary. A few cases, however, require more than that, and he is now more and more using chemical antiseptics. He had presented before this and other societies papers on that subject, and had presented facts in regard to the action of nitrate of silver. Also, about 15 years ago, he experimented with the phenol group, and with turpentines, bichloride, and other groups, but now he confines himself to the use of nitrate of silver. In the chronic cases he depends entirely on this drug. If we analyze the results obtained in other specialties, gynaecology, genito?urinary, opthalomology, we find that it is used very largely. In chronic gonorrhea, the pathologists tell us the germ is lodged deep in the membrane and in order to reach them some agent is required that will produce a superficial desquamation and nitrate of silver is the one agent they rely upon.

In chronic conjunctivitis the same pathalogic phenomona exist, and the opthomologist depends chiefly on solutions of nitrate of silver to obtain a radical cure. Those who had worked in the dead house had no doubt been impressed with the extreme thinness of the mucous membrane lining the various sinuses. Therefore, in chronic inflammation of these sinuses we find marked hyperplasia and a pyogenic infection.

If nitrate of silver has such a marked reaction in the other specialties, it appeared to him that the same rational reaction would occur in the nose. This led him to experiment with the drug; and the more he uses it the more greatly is he impressed with its value. He expressed regret that repeated presentation of the subject before this society had not induced the members to at least give it a thorough trial. Cures can be obtained in from three to six weeks providing all details of the technique are adhered to.

There is another point upon which he took issue with the reader of the paper, that is, the relationship of tooth infections to chronic empyema of the antrum. Dr. Babcock had stated that he hardly found a third of his cases due to tooth infection. Dr. Abraham was inclined to think that in his experiences the proportion of these cases was at least 50 per cent and probably greater. It is his rule now in all cases of chronic empyema in which nerve extraction of any molar has been resorted to, which has any relationship to its antrum, to have an X-ray taken immediately. It is remarkable to find the number of cases of root infection followed by an osteomyelitis and extension of the pathological process into the antrum. A considerable number of these cases do not present themselves to the specialist until an acute inflammatory process supervenes on the chronic lesion that is present. Therefore, one is liable to overlook the underlying tooth complications as a direct etiologic factor in the production of the chronic empyema. Considerable could be said about this interesting subject, but the time allowed prevents the covering of more ground.

Dr. MacKenty said that we are only on the edge of solving the problem of sinus disease; so many points are constantly coming up that it seems to be more difficult and obscure as we progress. The toxic cases are a problem in themselves, i. e., the cause of sinusitis with toxaemia. The degree of toxaemia is not always proportionate to the extent of sinus involvement. An almost negligible sphenoidal involvement may produce profound constitutional symptoms. Why it is so difficult in some cases to keep the sinuses open after operation? There must be in this type of case some concomitant bone involvement and a productive bone inflammation. The paper of the evening reflected great credit upon the reader, since it represented prolonged and painstaking observations.

Dr. Babcock, replying to Dr. Balls' suggestions in regard to the use of immune serum said that they hoped to be able to try it soon.

Dr. Dwyer's points were very well taken. The bacteria found may not be the inciting organisms, but recently he had become convinced of the connection between the pneumococcus and the "colds" where that organism is found. Many of the organisms found were doubtless secondary invaders.

The cures were determined by clinical examination only.

Dr. Flexner had brought up the point of finding different organisms or the same organisms at different times in the same attack. A few cases cultured late in an attack showed the same organisms that were found during the attack or no growth; but usually more than one culture was not taken for the same attack.

Dr. Abraham suggested that silver nitrate had proved very successful in his hands, but Dr. Babcock could not say anything about it from his own experience.

Finding several organisms in one case makes it hard to determine which is pathogenic in many cases, but when one organism is found with the frequency of the pneumococcus, it seems evident that they are at least associated with if not the entire cause of the diseases.

Referring to the pathogenicity of staphylococci of which Dr. Flexner spoke, Dr. Dwyer has brought out that point. Dr. Coakley wanted this subject investigated thoroughly and Dr. Zinsser was consulted as to the best way to do this. He thought it would require many months and many animals as different strains would have to be compared, and it was impossible to find time for that.

In reference to the matter of tooth infection, Dr. Babcock said they had carefully investigated that point and had used Radiographs freely and in cases where tooth infection was suspected capable dentists had been consulted, and in the few cases where tooth infection seemed a possible cause of antral trouble, the connection was not clear. Some men have proved bacteriologically that there is a connection. Turner and Lewis report that they found one-third of their cases due to tooth infection, but this was not borne out in the series just reported.

Case of Destructive Leishmaniosis. O. TORRES, *New Orleans Med. and Surg. Jour.*, Feb., 1918.

The patient, a woman of 56, had an inflammatory growth in the right side of the nose causing obstructed breathing on that side. A diagnosis of lupus was made and subsequently a diagnosis of lues. The lesion was never painful. Treatment in accordance with the diagnoses was of no avail. In the following year the ulceration appeared externally and continued progressively to descend until it reached the middle part of the nasal pyramid, destroying the entire lobule of the nose, then passing to the left side, leaving only a small portion, a strip of skin 3 or 4 m.m. long, which was all that remained of the left nostril. The latter destruction involved both nasal bones, the entire nasal pyramid being destroyed. There was total destruction of the septum as well as of the lower turbinates. Treatment with tartar emetic was instituted, even before the Leishman bodies were found, beginning with 0.05 gm. doses by injection and also the application of 1 to 1,000 tartar emetic solution to the ulcerations. The progress of the disease was arrested. ED.